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SYMPOSIUM.

WHAT CAN BE DONE FOR CHRONIC PROGRESSIVE DEAFNESS?

(a)—A STUDY IN THE TREATMENT OF DEAFNESS.*

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The purpose of this paper is to consider the aural pathology producing a chronic progressive deafness, to discuss the conservative methods of treating this condition, to compare the hearing of patients so treated over a period of years, and to evaluate the results of such treatment.

The material for this study comprises 200 cases under the personal observation of the author for an average of five years and 10 months, and represents patients in whom some improvement might be expected.

Only three factors have determined the selection of this particular group; namely, the completeness of the treatment, the length of time under observation, and the accuracy of the comparative examinations. The youngest patient was age 5 years; the oldest, age 78 years. In every instance the Wassermann was negative. The duration of deafness at the onset of the treatment varied from a few weeks to several years.

In all cases, the comparisons are made of the ability to hear spoken voice and whisper; and in all cases, except those

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antedating the audiometer, complete audiometric comparisons are made as well. The ability to hear a spoken voice and whisper at 20 feet (the United States Veterans' Facility standard) was considered normal.

In the audiometric comparisons, the regulation Western Electric audiometric formula was used. If deafness was present in both ears and the difference between the hearing in the two ears was not more than 20 dcb., the comparison in each case was based on the tests of the poorer ear.

The interval between these examinations was never less than six months, and in most instances several years.

Although a progressive deafness was the chief complaint in each instance, the following chart will illustrate the types of pathology producing the deafness and the frequency with which each type occurred.

CHART 1.

Types of Pathology Producing Deafness in 200 Cases.

1. Chronic Catarrhal Otitis Media	57.5%
2. Chronic Purulent Otitis Media	13.5%
3. Nerve Deafness	11. %
4. Otosclerosis	5. %
5. Chronic Purulent Otitis Media (one side); Chronic Catarrhal Otitis Media (one side)	5. %
6. Chronic Purulent Otitis Media (one side); Nerve Deafness (one side)	2. %
7. Chronic Purulent Otitis Media (one side); Radical Mastoid (one side)	1. %
8. Chronic Catarrhal Otitis Media (one side); Nerve Deafness (one side)	2. %
9. Chronic Catarrhal Otitis Media (one side); Radical Mastoid (one side)	1. %
10. Chronic Catarrhal Otitis Media (one side); Simple Mastoidectomy (one side)	1. %
11. Double Simple Mastoidectomy	.5%
12. Recurrent Acute Catarrhal Otitis Media	.5%
	Total, 100%

We have tabulated, then, various types and combinations of pathology producing a chronic progressive deafness. For the sake of comparison, however, we shall redivide these cases into four general classes; namely,

1. Chronic Catarrhal Otitis Media.

2. Chronic Purulent Otitis Media.

3. Nerve Deafness.

4. Otosclerosis.

The condition of the tympanic cavities in these cases may be summarized as follows:

CHART 2.

Condition of Tympanic Cavities in 200 Cases of Deafness.	
1. Normal Drumheads	58%
2. Retracted Drumheads	15%
3. Thickened Drumheads	1%
4. Chronic Purulent Otitis Media (active and inactive..)	26%
Total,	100%

The condition of the Eustachian tubes was as follows:

CHART 3.

Condition of Eustachian Tubes in 200 Cases of Deafness.	
1. Tubes always open	52%
2. Tubes partially to completely open	46%
3. Tubes persistently constricted	2%
Total,	100%

In attempting to restore the hearing of these patients to normal, the following otologic axioms were considered essential to acutely normal hearing:

CHART 4.

Otologic Axioms.
1. The external ear should be patent.
2. The drumhead should be normal in contexture and position.
3. For the drumhead to have its greatest receptive acuity it must be taut, and the air pressure on both sides must be equal.
4. To this end the Eustachian tube must be normally open.
5. The tympanic cavity must be filled with air at atmospheric pressure; it cannot contain serum, blood, pus, granulations, polypi, or cholesteatoma.
6. The ossicles must be mobile.
7. The internal ear nerve mechanism and cerebral centres must be functioning normally.

To the fulfillment of these conditions the course of treatment of these cases was directed. The treatment, then, assumed the following forms:

CHART 5.

Conservative Treatment of Deafness.

A. Correction of Infective Processes.
  1. Removal of granulations, polypi, cholesteatoma, etc.
  2. Supplementary local treatment.
  3. Simple or partial radical mastoidectomy.

B. Attempts to Restore to Normalcy the Conductive Mechanism in the Middle Ear.
  1. Inflation with or without Eustachian instillation.
  2. Eustachian Bougie.
  3. Otomassage.
  4. Correction of nasopharyngeal pathology.
    a. Removal of tonsils and adenoids.
    b. Removal of Eustachian or nasopharyngeal adhesions.
    c. Submucous resection and other intranasal procedures.
  5. Correction of temperomandibular joint displacement and dental malocclusion.
  6. Thermal procedures.
    a. Infrared
    b. Ultraviolet.
    c. Medical diathermy.
    d. Short wave therapy.

C. Attempts to Correct or Stimulate the Nerve Mechanism.
  1. Removal of all foci of infection.
  2. High frequency vibration.
  3. Phonographic exercises.
  4. Various strychnine tonics, etc.

D. General Treatment.
  1. Any procedure to improve the general health.
  2. Endocrine therapy.
  3. Vitamin B, etc.

A study of the periodical hearing tests on these 200 patients resulted in much statistical information, a part of which is here recorded. The following charts (see Chart 6, A and B) show the percentage of these patients whose hearing, under treatment, improved, became worse, or remained the same.

One of these charts indicates the audiometric interpretation of hearing; the other, the patient's interpretation; that is, his ability to hear spoken voice and whisper on a basis of 20/20.

CHART 6.

Percentage of Patients Whose Hearing Improved, Became Worse, or Remained the Same During Treatment and Observation.

Type	Better	Worse	Same
A—As Indicated by Audiometer.			
Chronic Catarrhal Otitis Media	52%	44%	4%
Chronic Purulent Otitis Media	47%	47%	6%
Nerve Deafness	25%	74%	1%
Otosclerosis	57%	42%	1%
Type	**Better**	**Worse**	**Same**
B—As Indicated by Ability to Hear Spoken Voice and Whisper.			
Chronic Catarrhal Otitis Media	51%	45%	4%
Chronic Purulent Otitis Media	57%	36%	7%
Nerve Deafness	23%	76%	1%
Otosclerosis	41%	58%	1%

The foregoing tables give some qualitative information concerning the results obtained from the conservative treatment of deafness. We shall now consider some quantitative aspects of the subject.

CHART 7.

The Amount of Gain or Loss in Hearing Sustained During Treatment or Observation.

Type	Hearing Improved		Hearing Became Worse	
	Average Gain	Greatest Gain	Average Loss	Greatest Loss
Chronic Catarrhal Otitis Media	7.3%	26%	9.4%	39%
Chronic Purulent Otitis Media	13. %	28%	9.9%	24%
Nerve Deafness	8. %	10%	14. %	40%
Otosclerosis	9.8%	16%	13. %	30%

The following charts tabulate further information concerning the amount of hearing gained or lost under treatment:

CHART 8.

Classification of Patients According to the Amount of Hearing Gained or Lost During Treatment or Observation as Indicated by the Audiometer.

Type	Gained over 20% Hearing	Gained over 10% Hearing
A—Patients Whose Hearing Improved.		
Chronic Catarrhal Otitis Media	.5% of the patients	25% of the patients
Chronic Purulent Otitis Media	25. % of the patients	60% of the patients
Nerve Deafness	(Greatest Gain, 10%)	
Otosclerosis	None	50%

Type	Lost over 20% Hearing	Lost over 10% Hearing
B—Patients Whose Hearing Became Worse.		
Chronic Catarrhal Otitis Media	9.5% of the patients	28% of the patients
Chronic Purulent Otitis Media	10. % of the patients	40% of the patients
Nerve Deafness	14. % of the patients	42% of the patients
Otosclerosis	50. % of the patients	

INCIDENTAL THOUGHTS AND OPINIONS.

1. This series has no controls, consequently there is no way to determine how many patients might have been improved without treatment.

2. Individual patients occasionally insisted that they heard better or worse; whereas, neither the ability to hear spoken voice or whisper, nor the audiometeric examination, substantiated the opinion.

3. The statement of certain patients that they hear better when the ear is running was occasionally verified. It seems to be true, however, that the dry ear usually functions better, although certain ears after being dry for a year or more lost function.

4. The more complete a radical mastoid operation, the greater the impairment of hearing.

5. Submucous resections did not improve the hearing in any of these patients.

6. Repeated Eustachian inflations may not result in opening the tube; whereas, a rest from inflation may result in spontaneous opening.

CONCLUSIONS.

1. Two hundred cases of chronic progressive deafness have been analyzed according to the underlying pathology producing deafness.

2. Various conservative methods have been employed in the treatment of this condition.

3. Accurate records have been kept of the hearing of these patients while under treatment or observation during an average of five years and 10 months, and a statistical study made.

4. Although the study indicates a result in the treatment of chronic progressive deafness that is not gratifying, it represents the best that we were able to accomplish and is our response to the demand of patients, not hopelessly deafened, to exert some effort in their behalf.

5. The present conservative treatment of chronic progressive deafness is unsatisfactory and inefficient.

Medical Arts Building.

SYMPOSIUM.

WHAT CAN BE DONE FOR CHRONIC PROGRESSIVE DEAFNESS?

(b)—RATIONALE, TECHNIQUE, CASE REPORTS AND OBSERVATIONS WITH GRAFTS IN THE ROUND WINDOW.*†

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A brief report of a small series of cases in which a tissue graft was placed in the round-window niche has already appeared.[1] The present discussion will serve to present additional case reports, two in which bilateral operations have been performed, minor modification of the technical procedure, an analysis of the immediate and late postoperative findings, and an elaboration of rationale of the procedure in the light of these observations.

At the very outset, one point of prime importance must be emphasized, which seems applicable, not only to the particular subject under discussion, but to any therapeutic measure, medical or surgical, employed for the alleviation of deafness. However essential properly taken audiograms of any patient's air and bone conduction may be, recent reports in the literature have shown that deafness itself may be but one manifestation of a variety of constitutional disorders. Treatment, therefore, of a single symptom is entirely improper unless all possible contributing factors have been taken into careful consideration. It is unnecessary to enumerate these many remote conditions which may be important contributing factors. Specific therapy should be used only after full knowledge of such possible influences is obtained and adequate treatment for their correction instituted. It is quite true that multiple therapy tends to obscure the clarity of analysis but the patient's interest demands that an exhaustive diagnostic study be made before any operative procedure should be attempted.

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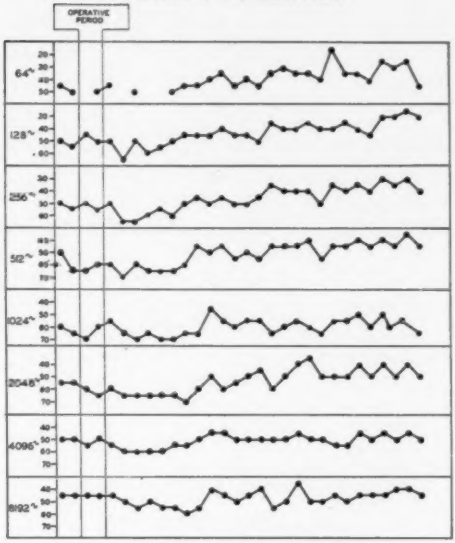
In the original report, definite standards of selection were outlined, which have subsequently been modified to a slight degree. In view of recent experimental and clinical findings, impairment of hearing, due in whole or in part to involvement of the neural mechanism of the ear, need not necessarily be a contraindication to operation.

The demonstrated extent of nerve involvement and its probable cause are alone the standards whereby operability can be determined. Routine loudness balances, repeated bone conduction audiograms with appropriate masking, supplemented by fatigue tests, give accurate information as to the amount of organ of Corti and nerve remaining. If, as has been hypothecated and demonstrated clinically, fixation of the round-window membrane increases the intensity of a given stimulus as delivered to the sensory receptors, the effect should be identical to increasing the actual intensity of the sound stimulus itself. This being the case, the nerve elements remaining would of necessity function more efficiently and in a manner analogous to the effect obtained in the variable type of loudness balance.

Advanced age and an impairment greater than 50 dcb. for the critical frequency range are still regarded as contraindications to successful surgery. An earlier statement that all other forms of treatment must have been tried and found unsuccessful is, in general, a phase of the situation which takes care of itself automatically. Few patients are referred for operation or come on their own initiative until long courses of routine treatment have proved unavailing, both in regard to improvement in hearing, or in serving to arrest further progress.

It is proper to assume that the less the impairment the greater is the possibility of benefit from any therapeutic procedure. At least, if improvement does result after an interval of time it will certainly be appreciable to such a patient and provide a more useful hearing level. In a few instances, where good bone conduction was found and recourse might always be had to a bone conduction hearing aid, the better of the two ears has been operated upon. If poor bone conduction is found, the poorer ear has always been operated upon first. When the ears reach the same level or the operated ear becomes the better of the two, a second operation may be

CASE 1—20 MONTHS.



CASE 2—NINE MONTHS.

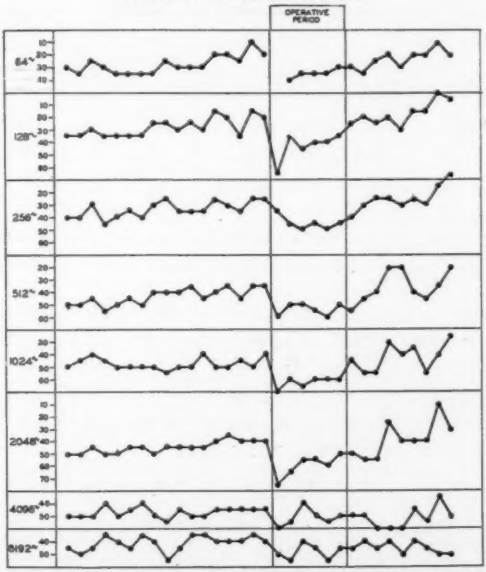


Chart 1. Composite audiogram of Case 1, which has been under observation for 20 months, showing gradual improvement. Case 2 is a composite audiogram of the right ear of Case 1, showing relatively fixed level of hearing loss with operative period 11 months after the operation upon the left ear. A continuing improvement is still shown nine months since this second operation.

performed. This has been done in two cases after a sufficient interval had elapsed to indicate that the improvement resulting from the original operation might reasonably be expected to be permanent. This interval of time has arbitrarily been set at one year (see Chart 1).

All patients, and particularly those who seem appropriate for operation, are given a thorough diagnostic study, consisting of repeated hearing tests of air and bone conduction, loudness balances, fatigue tests and careful analyses of the

CASE 18—17 DAYS.

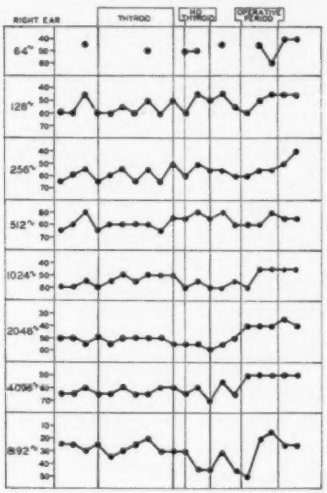


Chart 2. Hypothyroid, showing slight improvement during period of thyroid treatment, a return to original level when thyroid was discontinued, and immediate improvement following a round-window graft operation.

audiograms with reference to the degree of impairment of individual frequencies. The patient is concerned with loss of serviceable hearing, so the frequency range, 256 to 4,096 cycles, must be considered as most important from the therapeutic standpoint. Impairment of higher frequencies is of diagnostic and prognostic value alone.

The profound effect that remote influences may have upon impaired hearing makes a complete clinical survey an essential part of any diagnostic study. The cardiovascular system, the glandular system, blood chemistry and serology, evaluation of

nutritional and allergic disturbances must all be investigated and corrected if necessary. The elimination of remote disorders may well bring about a 10 dcb. improvement in the hearing level before any specific measures are undertaken (see Chart 2).

Any special operative technique must of necessity be a matter of gradual refinement and development. Successful placing of a tissue graft in the round-window niche is essentially not a difficult procedure, provided certain points are borne in mind. Control of bleeding is of prime importance. Painstaking and gentle cleansing of the external canal and outer surface of the tympanic membrane will more than make up for the time consumed by obviating the inevitable hyperemia of the tissues resulting from too vigorous cleansing. Adrenaline, though extremely useful, must be used circumspectly to avoid too great absorption through the round-window membrane. The prone position lends itself most favorably to ease of inspection and manipulation, while any serum that may accumulate will not be in the neighborhood of the round window. The incision of the tympanic membrane must be of such size as to afford complete inspection of the posterior half of the middle ear cavity. Careful placement of the graft in the niche and firm packing without damage to the round-window membrane is essential. Magnification and brilliant illumination, though perhaps not essential, certainly add to the facility with which the operation can be performed and permits the actual operative procedure to be carried out in approximately half an hour. The postoperative course has been quite uneventful and the patient may leave the hospital on the third or fourth day, when the first dressing is done. Until the drum has healed, the most scrupulous technique must be observed in doing the fairly frequent dressings. The variation in the length of time required for complete healing to occur is difficult to explain and is apparently not dependent upon the size of the opening or the preoperative condition of the drum. A pin-point opening may persist for a week or 10 days before final complete closure occurs. The opening closes in any period from one week to a month.

During the immediate postoperative period, audiograms are made every three days and at least once a week for several months thereafter. Subsequently, the patients are seen every

month and careful examinations made. The audiograms are, of course, made of both ears. Repeated audiometric examinations tend to increase the patient's accuracy of response but do not increase the actual acuity of hearing. In all preliminary studies at least three such examinations are made to obtain a true picture of the patient's real hearing loss. When subjective improvement occurs, the patient is questioned carefully as to the exact changes in hearing which have been noticed. Sounds of different pitch observed for the first time, uncomfortable sensations aroused by sounds of high intensity, ability to use the telephone again, increased ease of participating in group conversation, perception of remote sounds, may all be accepted as actual improvement. *Vis a vis* conversations are of no importance as a test of improvement, as every deaf person who has been so disabled for an appreciable length of time reads lips, whether consciously or not. An imagined improvement observed in this manner will disappear in a darkened room. In defense of audiometric examinations as a measure of improved hearing, it must be said that where there is a true subjective increase in the hearing level this will always be reflected in the threshold audiograms. The reverse is not necessarily the case. In the last analysis, intelligibility of speech must be the standard of improvement, and, as a corollary, improvemnt of the patient's own voice and speech must necessarily follow. Such voice recordings are interesting and important in the final analysis of postoperative results. Every possible precaution must be taken to avoid contributing to the extraordinary euphoria which is so much a part of every deafened person's reaction to any form of therapy.

A round-window graft has been placed in 25 ears. In two cases, the bilateral operation has been done as stated above. Fascia alone, fat and fascia, or periosteum have been used. The tissue is obtained through a small incision made immediately behind the base of the ear. With the technique of implanting which has been previously described the graft has never failed to take. It can be observed through the drum opening as it first becomes pale and later assumes the blush of beginning vascularization, a picture familiar to anyone with experience in tissue-grafting elsewhere in the body. In some of the cases improvement has been delayed for from three to as much as seven months; in others, improvement occurs while the drum is still open. In no instance has the

hearing in the operated ear failed to return to its original level, while in 90 per cent of the cases hearing has been improved at least 10 dcb. for the critical frequencies and this improvement maintained during the period of observation. In 60 per cent of the cases, the poorer ear has reached or surpassed the level of the better ear. In 50 per cent of the cases, the impairment of the better ear has progressed, while the hearing level in the operated ear either remained stationary or continued its improvement. Where no significant improvement has taken place, the hearing in the operated ear has been fixed at its preoperative level. In no case has any other specific treatment been employed.

Adequately to rationalize this procedure, an analysis has been made of each individual ear in regard to age, sex, clinical findings, otoscopic examination, preoperative hearing level in general, with reference to individual frequencies, and the immediate and late postoperative reaction with a second analysis of the audiograms.

These patients have two things in common: a wide incision of the drum and a tissue graft placed in the round-window niche. The observed middle-ear pathology has been distinct in every instance. The postoperative course has been largely individual. From the earliest to the most recent case, the tendency to improvement has been constant and sustained. Myringotomy alone in markedly relaxed or retracted drums may bring about a temporary but unstable improvement while the sustained effect of this operative procedure permits of no other ready explanation than the fact that the round-window niche has been obliterated and the resilient round-window membrane fixed.

An analysis of these operative cases follows. At the very outset, one prerequisite must be accepted. If any surgical treatment of deafness is justified, it must expose the patient to a minimum of risk and offer a reasonable hope of useful improvement. This surgical effort must be balanced against the established worth of the electrical hearing aid, which, when intelligently prescribed by a physician familiar with the intricacies of the problem, immediately solves the difficulties of a large group of deafened individuals. This is particularly true when bone conduction remains within reasonably normal limits.

An impairment of hearing involving a loss of 40 to 50 dcb. in the critical frequencies can probably never be cured, in the sense that the individual's hearing will return to normal. In this discussion the terms "impairment" and "normal" are predicated on objective measurements of threshold hearing against an established normal level. It must be assumed that mechanical and human error are eliminated from the picture within all reasonable limits. Standardization of technique and equipment is taken for granted. In the present instance, constant check against inaccurate findings has been carried out with the same precautions used in any well controlled experimental procedure.

The general feeling that threshold response is not necessarily an indication of serviceable hearing is undoubtedly true, but a change in the hearing level of + or —10 dcb., maintained for any considerable period of time, is a clear-cut indication that the particular ear is either improving or becoming further impaired. The term "serviceable" is used in relation to the individual's total hearing, a binaural phenomenon, which it is reasonable to assume represents in the cortex different frequencies perceived at a high level in either ear. When ears are impaired almost equally, a 10 or 15 dcb. greater loss in one of the critical frequencies in one ear will render that particular ear unserviceable for individual use.

In this series of cases none is discussed which has not been observed for at least one month postoperatively, and only two less than three months. The longest period of observation since operation is 20 months. All patients have previously received adequate nose and throat care and all have had routine measures employed for the relief of deafness without success. In addition, many, if not all, of the cases have undergone some of the more bizarre types of treatment, which are unfortunately readily available at the moment.

A brief resumé of the otoscopic examinations of the tympanic membranes follows:

Case 1: Considerably thickened and retracted; light reflex normal; edge of niche seen through drum.

Case 2: Thickened and retracted; light reflex absent.

Case 3: Thickened in lower posterior quadrant; no retraction; light reflex normal.

Case 4: Slightly thickened and retracted; light reflex normal.

Case 5: Slightly thickened and retracted; light reflex normal.

Case 6: Very much retracted, moderate thickening; light reflex normal.

Case 7: Slightly thickened and retracted; light reflex normal.

Case 8: Slightly thickened; no retraction; sclerotic area lower part; light reflex normal.

Case 9: Slightly thickened; no retraction; sclerotic area lower part; light reflex normal; impossible to see niche.

Case 10: Thickened and retracted; light reflex normal.

Case 11: Moderately retracted but membrane quite delicate; light reflex normal.

Case 12: Markedly thickened and retracted, with irregularity of surface posteriorly; light reflex absent.

Case 13: Membrane quite clear, with some thickening along posterior margin; light reflex absent.

Case 14: Slightly thickened, with moderate retraction, particularly anterior; light reflex reduced.

Case 15: Markedly thickened and retracted; light reflex reduced.

Case 16: Membrane retracted and thickened and dull in appearance; promontory normal color; light reflex normal.

Case 17: Markedly thickened and retracted; probably adherent; light reflex absent.

Case 18: Retracted anteriorly; posterior half normal in appearance; light reflex normal.

It will be noted that, whereas the majority of the cases exhibit retraction and thickening of the membrane, in at least half, these changes are not in themselves sufficient to account entirely for the impairment in hearing.

The following classification details the sex, age, percentage preoperative impairment, percentage improvement postoperative, the effect upon the opposite ear, and the subjective report of improvement. It has been observed rather frequently that when a sudden marked improvement occurs in the operated ear this change may be reflected in the other ear. Permanent improvement of this character in the unoperated ear has not been found (see Table I).

TABLE I

CASE NO.	SEX	AGE	EAR	PREOPERATIVE IMPAIRMENT	BC	POSTOPERATIVE IMPROVEMENT	OPPOSITE EAR IMPROVED	SUBJECTIVE IMPROVEMENT
1	F	17	R	49%	<AC	20%	–	+ + +
2	F	17	L	31%	<AC	20%	–	+ + +
3	F	45	R	40%	=AC	0%	–	+
4	F	21	R	41%	>AC	10%	–	+ +
5	F	21	L	32%	=AC	5%	–	+
6	F	26	R	46%	>AC	8%	–	+
7	F	24	R	43%	>AC	8%	–	+
8	M	29	L	36%	>AC	8%	–	–
9	F	35	R	57%	<AC	5%	–	–
10	F	42	R	48%	=AC	8%	–	+ +
11	F	39	R	33%	<AC	13%	+	+
12	F	44	L	48%	=AC	24%	–	+
13	F	54	L	40%	<AC	0%	–	–
14	F	47	L	43%	<AC	0%	–	–
15	F	41	R	37%	<AC	13%	–	+
16	M	41	L	39%	=AC	0%	–	–
17	M	18	R	37%	>AC	6%	–	–
18	M	35	R	46%	>AC	10%	–	–

TABLE II

CASE NO.	EAR	IMMEDIATE EFFECT OF DRUM OPEN	IMMEDIATE EFFECT OF DRUM HEALED	DURATION OF POSTOPERATIVE IMPAIRMENT	BEGINNING OF POSTOPERATIVE IMPROVEMENT
				MONTHS	MONTHS
1	L	0	+	4	7
2	R	–	+	1	3
3	L	+	+		2
4	L	–	0		1
5	R	0	0		5
6	R	0	+		3
7	R	–	0		5
8	L	–	0		4
9	R	–	+		1
10	R	–	+		1
11	R	–	0	1	2
12	L	–	0		1
13	L	–	–	2	
14	L	–	0	2	3
15	R	–	–		1½
16	L	–	0	2	
17	R	–	+		IMMEDIATE
18	R	–	+		IMMEDIATE

In this particular analysis, the percentage hearing loss is used simply to satisfy the general demand for such an estimation. Under percentage improvement, no true idea can be obtained of the benefit derived since a sustained loss at 2,048 cycles would more than offset a striking improvement of 512 cycles, and *vice versa*. Subjective improvement is measured in terms of new sounds heard.

During the operative period, or interval between actual operation and healing of the drum, either impairment or improvement may occur. In most instances, there is an impairment of the lower frequencies and slight improvement of the high. At the moment of healing, this situation usually reverses itself. If impairment is present it may persist for a short while. Real improvement, as in the first case, did not appear for seven months. The following tabulation details these responses (see Table II).

TABLE III

POSTOPERATIVE CHANGE

CASE NO.	MONTHS SINCE OPERATION	EARLY								LATE							
		64	128	256	512	1024	2048	4096	8192	64	128	256	512	1024	2048	4096	8192
1	20	–	–	–	0	–	–	–	0	+	+	+	+	+	+	+	0
2	9	–	–	0	0	0	–	0	–	+	+	+	+	+	+	+	+
3	16	+	+	+	0	0	0	+	0	0	0	0	0	0	0	0	+
4	19	0	0	0	0	0	+	0		+	+	+	+	0	+	+	
5	8	0	0	0	0	0	0	0	0	+	+	+	0	0	0	+	0
6	12	0	–	0	0	0	0	0	0	0	+	+	+	+	0	+	0
7	14	0	0	0	0	0	0	0	0	+	+	+	0	+	+	0	0
8	12	0	0	0	0	0	0	0	0	0	0	+	+	+	0	0	0
9	14	0	0	0	–	–	–	0	0	+	+	+	+	+	+	0	0
10	12	+	+	+	+	+	0	0	–	+	+	+	+	+	+	0	0
11	4	0	0	0	0	0	0	0	–	+	+	+	+	+	+	+	0
12	6	0	0	0	0	0	0	0	0	+	+	+	0	+	+	0	0
13	7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
14	4	0	0	0	0	0	0	0	0	+	+	+	0	+	+	0	+
15	5	0	0	0	0	0	0	0	0	+	+	+	+	0	+	0	0
16	3	0	0	–	0	0	–	–	0	0	0	0	0	0	0	0	0
17	1	0	0	0	0	–	–	0	0	+	+	0	+	+	0	+	+
18	1	+	+	0	+	+	+	+	0	+	+	0	+	+	+	+	0

Early and late postoperative improvement for individual frequencies is of interest in such an analysis and it can be seen from the following that when improvement did occur, in the great majority of cases it was late, and that when it occurred early, the gain persisted (see Table III).

From the point of view of a useful improvement, interest centers upon the frequencies which have been affected favorably most often and to the greatest extent. It can readily be seen that improvement has occurred more often in the critical frequencies, 256 to 2,048 cycles, than in the extremes of the frequency range, and that the most marked improvement for any individual frequency has been in this same range. The average improvement for 13 cases for all frequencies in decibels is shown below. In one of these cases, no improvement at all has developed after six months. In two others, only a single frequency has shown any change. Where no change whatever has resulted, the zero has been counted in arriving at the average. In no instance has further impairment of the preoperative level developed (see Table IV).

DISCUSSION.

The foregoing data is derived from a critical and controlled study of the effect of blocking the round-window niche of deafened ears with a tissue graft. Many of the observed effects of this procedure are still unexplained, but the general uniformity of results and the consistent but gradual improvement justifies its further employment. The clinical response does not conform exactly to that observed in experimental procedures and, hence, does not permit of all of the explanations advanced for these reported phenomena. In the light of the very dramatic and striking results of surgery of the labyrinth which are being reported at the present time, fixa-

TABLE IV

POSTOPERATIVE IMPROVEMENT IN DECIBELS

CASE NO.	64	128	256	512	1024	2048	4096	8192
1	10	30	35	30	25	25	10	0
2	10	20	30	35	35	20	10	0
3	5	5	0	0	10	0	0	0
4	15	5	5	10	0	10	10	0
5	15	10	15	10	0	15	5	0
6	0	0	10	0	15	0	30	5
7	10	15	20	5	5	10	0	0
8	0	0	10	10	5	0	5	0
9	5	5	5	5	0	5	0	0
10	10	10	5	10	0	20	5	0
11	15	5	5	10	0	20	15	0
12	5	0	10	0	10	15	0	0
13	0	0	0	0	5	0	0	0
14	5	5	5	0	10	15	0	5
15		20	15	20	10	20	0	0
17	5	5	0	10	10	10	10	30
18	15	15	20	5	10	10	10	15
AVERAGE	8	9	11	10	10	12	6	4
MAXIMUM	15	30	35	35	35	25	30	30
NO. OF CASES WITH IMPROVEMENT OF 10 DECIBELS OR MORE	8	7	9	9	9	12	7	2

tion of the round-window membrane might well seem definitely contraindicated as a method of treating deafness; however, the clinical observations of the effect of acutely packing the niche with cotton in patients with large perforations of the drum and the data reported here are statements of observed fact. There is no reason at the present time to modify the explanation already advanced, that fixation of the round-window membrane seems to eliminate its natural damping effect upon vibration and possibly affords additional reflecting surface. The late appearance of favorable response must be due to the gradual tightening of the graft as it adapts itself to the contour of the niche. Variations in accessibility due to the

exposure through the tympanic membrane, and individual variations of position of the niche, with consequent ease or difficulty in placing the graft, doubtless explains the differences in time at which the improvement first occurs.

CONCLUSIONS.

1. Twenty-five operations for blocking the round-window niche with a tissue graft as a treatment of deafness are reported. In two cases both ears have been operated upon. Eighteen of these cases have been observed postoperatively for at least three months, with the exception of two which represent a one-month interval. The longest period of observation has been 20 months.

2. In no case has the hearing been further impaired by operation. Two cases have shown no improvement whatever. All the other cases have shown improvement to an appreciable degree.

3. An analysis of the improvement obtained shows a maximum of 20 per cent in one patient. Improvement of 10 dcb. or more occurs most often in the frequency range, 256 to 4,096 cycles. The average change observed for all frequencies in all cases analyzed shows an increase of approximately 10 dcb. for all critical frequencies. This average has been made by including individual case frequencies in which no improvement occurred.

4. While subjective improvement has been accepted when representing an actual gain in useful hearing, threshold audiograms are the standard measure of change in all the cases here analyzed.

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Abington Memorial Hospital.

DISCUSSION.

DR. D. E. STAUNTON WISHART (Toronto): This paper by Dr. Hughson is the last in a series of four papers which follow one another in logical sequence and to each of which reference must be made if we are to discuss the subject adequately. These are:

1. Function of the Round Window, by Hughson and Crowe, 1931.

2. Immobilization of the Round-Window Membrane: A Further Experimental Study, by Hughson and Crowe, 1932.

3. Grafts in the Round Window in the Treatment of Certain Types of Deafness, by Hughson, 1937.

4. Today's paper.

The first pair deal with the underlying experimental data and the second pair with the results obtained on patients.

I am going to suggest, 1. that there is no adequate statement of the kind of deafness for which this round window operation is recommended; 2. a doubt that there is experimental evidence that good effects should be expected from this operation; and 3. a doubt that the good effects of the operation are of consequence.

1. The only statement Dr. Hughson made in paper 3, regarding the type of deafness suitable for his operation was: "The deafness cannot be of the nerve type as proved by adequate bone-conduction audiogram." He indicated the patient's hearing only with air conduction audiograms. But he gave no data regarding voice, whisper, tuning fork, 4A Audiometer or bone-conduction tests.

In paper 4, he suggests how to discover "accurate information as to the amount of organ of Corti and nerve remaining," but he fails to provide us with an accurate diagnosis of the kind, degree and cause of the deafness of the 18 cases he reports.

Several air-conduction audiograms were made before operation by the same observer with a machine kept up to standard at all times, in a quiet room and in a constant position. As far as the audiometer is concerned, his testing leaves nothing to be desired. He has also, with the aid of Dr. Witting, devised an ingenious apparatus to record "loudness balance." From personal observation I am convinced that his bone conduction tests are well and adequately performed.

After all, what we and the patient are interested in is not the patient's ability to hear a tone reproduced through a telephone receiver, but his ability to hear the spoken voice. It is common knowledge that some patients' appreciation of the spoken voice is better than the audiometer record would lead us to believe, and that other patients' hearing for the spoken voice is actually worse than the audiometer record would lead us to believe. It is my opinion, therefore, that his audiometer tests leave the practical otologist dissatisfied. He requires to know the patient's hearing as recorded by the other methods I have suggested as well as by the audiometer.

2. In paper 1, the results of acute experimental fixation of the membrane of the round window in animals were reported. Filling the niche of the round window with wax or plaster has no appreciable effect on the transmission of sound, but if the membrane of the round window was made rigid by pressing on it with a plug of moist cotton the perception of spoken words and practically all tones was increased at least 50 per cent.

These observations should be continuously borne in mind by the Society in evaluating Dr. Hughson's present contribution. For in paper 4, he states "if, as has been hypothecated and demonstrated clinically, fixation of the round window increases the intensity of a given stimulus as delivered to the sensory receptors, the effect should be identical to increasing the actual intensity of the sound stimulus itself."

This statement is based on two inferences: 1. That the Wever-Bray technique is a true index of the acuity of hearing, and 2. that for the operation to prove effectual it must produce on the round window permanent pressure.

I submit the Wever-Bray phenomenon may have something to do with hearing or, on the other hand, it may have nothing at all to do with it. Dr. Hughson's own opinion (page 206 of the 1937 Transactions of the American Otological Society) as given last year to this Society was: "I do not believe that any conclusive evidence has been advanced so far to prove that the two phenomena (cochlear response and actual hearing) are necessarily associated with each other."

In paper 2, four photomicrographs show the positions of grafts that were placed in the round window of experimental animals. In two of these a space is shown existing between the inner surface of the graft and the membrane of the round window. Inasmuch as improved transmission of sound was again demonstrated in all these cases, it has been assumed by the authors that the grafts were continuing to exert a pressure upon the membrane of the round window. I submit that these conclusions drawn from those photomicrographs are not convincing to a lay reader like myself. They suggest rather, that a plug has been successfully placed in the niche of the round window; that it is impossible to guarantee that such a plug will completely fill the niche right down to the window; and consequently it is unlikely that continuous pressure will be exerted subsequent to operation. Therefore, to my mind it suggests that the increased transmission of sound is capable of some other explanation than the one that is given.

I have purposely dwelt on these points in a sketchy fashion, for I have neither the information nor the knowledge necessary to discuss them. I hope that some of those in the audience who are thoroughly capable of answering these criticisms will do so or that some of the members of the Otological Society will demand an adequate explanation from those who are capable of giving it.

Human beings are not the same as experimental animals. Granted that a phenomenon holds for the experimental animal, it is no guarantee that the same phenomenon will hold for the human being.

The niche of the round window in the cat is an aperture which readily allows a direct observation of the round window. In the human it is otherwise. The niche of the round window leads into a right-angled bay. There are variations. Some of these bays are more accessible to packing than others, but again I am doubtful whether it is possible in the human being in more than a very small percentage of cases to insert a graft in the niche which will fill the angular channel and adequately cover the round-window membrane.

The experiments on the animals showed that in 50 per cent of the cases there was an air space between the graft and the membrane of the round window. The likelihood is that the incidence of such a result in man will be greater.

Therefore, a graft placed in the niche of the round window in a human being will be a plug and cannot exert pressure, although it must be admitted that it may exert a dampening effect by virtue of the closed column of air between graft and window. If, therefore, we follow the experimental facts upon which this operation has been founded, we will draw the conclusion that this operation—I quote from the original experimental paper—will result in the filling of the niche with a plug which "will have an appreciable effect on the transmission of sound."

3. This criticism so far has been purely theoretical. Dr. Hughson, however, placed in our hands 12 months ago a very detailed account of eight cases. Today he has added to this record that of 18 more. I have not had the details of the latter long enough to give them thorough study, but I have carefully studied the eight cases with which you are all familiar.

I saw Dr. Hughson perform two operations, and was amazed at the brilliant illumination and magnification which his binocular head magnifier

affords. The operation is performed as he describes and his technique leaves nothing to be desired. The section of drum cut away is large and Dr. Hughson assures me that it sloughs and that the opening closes by the gradual replacement of the drum from the cut edges. I saw two patients in whom the opening had closed. In one the new membrane was rough, slightly thickened and marked with fine blood vessels—and one where the new section of drum was only slightly thicker and less transparent than the rest of the drum. I have every confidence that the healing of the drum proceeds as he has described. I saw the audiometer tests performed upon these two patients and have no criticism, but only praise to offer.

But—what follows is a criticism of Dr. Hughson's results based on the facts presented in his own audiograms.

The six charts of paper 3 give data regarding 91 audiometer tests on the unoperated ear. The variations for each tone have been recorded and averaged (lantern slide). These show that Dr. Hughson, under ideal conditions, has demonstrated a variation in the audiometer record of each tone of between 10 and 15 dcb. The charting of the variation in the unoperated ear of Chart 1, Case 1, illustrates this nicely (lantern slide).

This variation corresponds with my own experience so that I feel no significance can be placed upon a variation of 5 dcb. in a record and not much more in an alteration of 10 dcb.

In today's paper, Dr. Hughson claims that "in 90 per cent of the cases hearing has been improved at least 10 dcb. for the critical frequencies." His ingenious method of massing a series of audiometer tests suggests improvement in hearing, but the Otological Society will understand the actual change when the alteration is presented in the ordinary form of audiometer chart. In the records I have recharted, a circle represents the right ear and a cross represents the left. I have used black ink to represent the audiogram of the best hearing prior to the operation and red ink to represent the hearing on the last date on which a record was made.

In Chart 1, Case 1, a comparison between the record before operation and that when last examined (lantern slide) shows a slight improvement in the hearing of five of the eight notes and a slight impairment in two. Neither the improvement nor the impairment was greater than 10 dcb. This is no greater than the variation which we feel to be normal and which was normal according to Dr. Hughson. You will see, however, that I have tried to be generous and have called this a demonstration that the patient's hearing was "improved."

I have similarly gone over all the charts of paper 3 and for your convenience have recharted them in the conventional manner (lantern slide). You will note that I have commented that Cases 1, 2 and 8 were "improved"; that Cases 4 and 6 were "unchanged," and that Case 5 was "worse." This I feel is a generous view of the result. A more critical evaluation, taking into consideration the variation established by Dr. Hughson on the unoperated ears, would suggest that the hearing in the entire group has been essentially unchanged.

But our analysis of the published cases can go farther. I have made a similar analysis of the records of the unoperated ears (lantern slide). I had some difficulty in classifying Case 2 and Case 6, but finally decided to call the first "improved" and the second one "unchanged." The result has been that I have classified two as "improved." three as "unchanged," and one as "worse."

A comparison of the judgments on the unoperated and operated ears (lantern slide) shows that both the operated and unoperated ears were improved in Cases 1 and 2, and that both ears were worse in Case 5, and that both were unchanged in Case 4 and Case 8, leaving the only discrepancy in Case 6, where the operated ear was improved and the unoperated ear was unchanged.

The correspondence suggests that the state of the patient's hearing was the result of the general condition of the patient or of her mental outlook rather than an effect of the operation.

Today's paper contains two tables (II and III), where the change in hearing recorded in Table II is, for certain patients, grossly at variance with that recorded in Table III.

What the fate of the graft placed in the round window ultimately is in Dr. Hughson's patients we have no means of knowing. Because of the great lack of any marked change one way or the other subsequent to operation, two conclusions are possible—either, first, that the graft disappears, leaving the round window functioning as before operation, or second, that if the graft remains as a plug, the round window has very little function. I submit that these conclusions are as possible as the very different conclusions placed upon his results by Dr. Hughson.

To quote again from paper 3. It is stated "that deafness alone does not warrant major surgical operation as a therapeutic measure; . . . however distressing the condition may be, its correction must not involve risk of life." Once again I must pay tribute to Dr. Hughson's technique. In his hands this procedure has proven safe, but I submit that the procedure is fraught with the danger of subsequent otitis media, labyrinthitis and meningitis. Further, it is stated that "there should not be more than an outside chance of further impairment." The results I have demonstrated to you do not convince me that improvement of consequence has followed. I suggest that the test that we demand is a production of records to show that some patients subsequent to this operation have been able to follow an occupation from which deafness had previously barred them. Severely deaf people will try anything once in an endeavor to improve their lot and for a long time subsequent to the taking up of a new procedure or a new apparatus or following an operation—hope, or whatever you call it, may buoy the patient up to a conviction that he hears better than before. Time, however, proves all things and I suggest that in most patients an improvement no greater than that shown in the attached record is more likely to produce an unfavorable psychological effect upon the patient.

In the concluding paragraph of this paper it is stated that "subjective improvement," though present in five cases, is too unreliable to be regarded as an actual measure of success." That is quite true when the subjective improvement is slight. Subjective improvement enough to allow the patient to follow an occupation previously prevented by the deafness is a different matter entirely. Dr. Shambaugh summed this up recently when he stated "the degree of deafness as determined by the audiometer does not give even an approximate idea of the difficulty the patient may have in hearing the voice. It is the handicap for the voice that really counts."

It will be noted that my criticisms are based on theories and facts presented in Dr. Hughson's actual papers. Nothing but praise must be given to Dr. Hughson for having provided us with the ammunition with which to meet his sponsorship for an operative procedure to improve hearing. It is a fault which other workers would do well to copy.

While I have nothing but admiration for the way in which Dr. Hughson has conducted and reported his procedure, it is my opinion that the results he has obtained so far will not justify other otologists to take up the operation as a likely means of improving hearing. Dr. Hughson works under such excellent conditions in such a truly scientific spirit and has reported his work in such admirable fashion that the further elucidation of this problem can be left in no better hands.

DR. WALTER HUGHSON (closing): I shall start as Dr. Wishart ended and say that I have nothing but the most complete admiration for the pains he has taken in preparing this discussion of my paper, and also for the conclusions he has reached, however adverse they may be to the therapeutic procedure under discussion. I do regret that his remarks have been confined

to a criticism of early experimental findings and a preliminary clinical report rather than to a discussion of the present paper, representing, as it does, a critical statistical analysis of 18 operative cases. The facts reported this morning are a matter of record and require no theoretical interpretation.

As we stated in our earlier paper, the operation is applicable to any case of conductive deafness in a nonsuppurating ear with an intact drum and in which the middle-ear cavity is not obliterated. The original statement that nerve deafness is a contraindication has now been modified as outlined in the present report.

When first performed, the operation was based on experimental findings in animals. The electrical response to acoustic stimulation did increase immediately, particularly for the higher frequencies. This phenomenon was observed repeatedly and subjected to the most rigid experimental control. For some obscure reason, the response to fixation of the round-window membrane in human beings does not conform, in the matter of time at any rate, to these experimental findings. We have previously expressed the conviction that the cochlear response in animals is not necessarily an index of subjective hearing. It is, however, a measure of the effect of experimentally produced middle- or inner-ear pathology.

It is obviously impossible in so short a space of time to comply with Dr. Wishart's desire that we present case histories even of so small a series of patients. Suffice it to say that every case has been subjected elsewhere to routine courses of inflation and bouginage, in some instances over long periods of time, but in no instance with any beneficial effect. Every case, whether old or young, has been at some time diagnosed otosclerosis, a term which is rapidly becoming applied rather indiscriminately to any conductive deafness. If a round-window graft were placed successfully in the niche of a patient suffering from otosclerosis that patient should theoretically be rendered completely deaf. Every graft has remained in place; in no instance has any individual's deafness been further impaired. By inference, we must assume that no patient had otosclerosis.

There is no more treacherous ground, in the whole field of otology, to tread upon than the realm of subjective improvement. True improvement of this sort has already been defined. It cannot be included in an analysis of this sort on any other basis. We prefer to discourage our patients until this subjective improvement has become a matter of course rather than a sensational experience of the moment.

The photomicrographs which Dr. Wishart has studied were made of sections of the temporal bones of animals sacrificed from 10 days to seven weeks after operation. They had all taken, he admits, but such tissue grafts contract slowly. Unfortunately for this particular discussion only, we cannot demonstrate the fate of the graft in our oldest case, 20 months. Dr. Wishart labors under a misapprehension in stating that these grafts increase intralabyrinthine pressure. Such a statement has never been made. The round-window membrane is fixed; the reflecting surface of the cochlea is increased, the latter a suggestion offered by Pohlman seven years ago.

Our composite audiograms present a consecutive picture of changes in threshold acuity heretofore unavailable to the otologists. Variations of + or —5 dcb., a possible total of 10 dcb., as Dr. Wishart points out, are of no significance; however, a consecutive and consistent improvement of from 5 to 10 dcb. is a matter of some consequence. It is true that the unoperated ear in most of these reported cases has shown variation. Dr. Wishart has analyzed Case 1, reported in our original paper. Compare the audiograms, Charts 1 and 2, herewith shown, and the consistent trend towards improvement postoperatively can scarcely be gainsaid.

Percentage improvement, average improvement of all frequencies, is a matter of no possible importance so far as actual hearing gained is concerned.

We are interested, as is the patient, in individual frequencies. A 10 dcb. gain at 2,048 cycles would more than offset a similar loss of both 256 and 128 cycles. Such a specifically critical view must be taken. Even in so small a series of cases, an average gain of 10 dcb., with extremes of zero to 30 dcb., must be accepted as a definite average improvement. We are not familiar with any other comparable results for any form of therapy.

Dr. Wishart felt that we were not quite living up to our original statement that no procedure for deafness should involve danger to the patient. It is quite true that this operation, placing a graft in the round-window niche, exposes the patient to the risk of infection, but we have not had an infection. Eustachian tube manipulation of any sort, I think, exposes the patient to as great, if not greater, risk of middle-ear infection as opening the drum and placing a graft in the round-window niche. Certain it is that such infections do occur.

Every deaf person presents a difficult psychological problem. In our experience, this has often been made critical by years of futile routine treatment. The data herewith presented makes it possible to give to individuals suffering from this disability a reasonable assurance that a substantial improvement may be hoped for and that further progress of impairment may be arrested. In Dr. Wishart's words, "I submit" that continued use of this particular form of treatment under the definitely restricted stipulations of selection more than warrants its employment.

SYMPOSIUM.

WHAT CAN BE DONE FOR CHRONIC PROGRESSIVE DEAFNESS?

(d)—SURGERY OF THE NASOPHARYNX IN THE TREATMENT OF CHRONIC PROGRESSIVE DEAFNESS.*

DR. HAROLD WALKER, Boston.

It requires a certain courage to discuss any problem which has to do with the treatment of chronic progressive loss of hearing, but it is only to be expected that over a period of 35 years, one's close association with a rather large number of persons suffering from decreased hearing must necessarily have resulted in certain observations and arrived at certain justifiable conclusions regarding the affection. In the first place, I have been impressed with the difficulty in making a diagnosis of the common forms of the disease which we call progressive deafness. If we eliminate the cases of decreased hearing due to suppuration, etc., then we are able to classify nearly all of them under two broad general heads: *1.* Otosclerosis; *2.* chronic adhesive inflammation of the middle ear, due to nasopharyngeal and other infections.

This latter class has been given many and varied names in the textbooks, but, to me, the pathological changes are what should suggest the proper nomenclature; and again, that is difficult because of the twofold widely differing results of the processes, namely, a slowly progressing fibrous change in the middle ear and at the same time a slow progressive atrophy of certain elements in the organ of Corti and ganglion cells. For a moment I shall refer to the first class and then dismiss it. *1.* Otosclerosis; it may be that I have been unfortunately "brought up" or too conservatively so, but I confess that I have difficulty in making such a diagnosis unless I have before me the clearly cut picture of Politzer. To me it shows a normal tympanic membrane generally with a faint blush over the promontory; then, objectively, a perfectly nor-

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mal nose and throat, good breathing space, freedom from colds, no variation in hearing except from fatigue, and an unobstructed and a normal functioning of the Eustachian tube. The patient, rather young, with a slight change in the hearing, beginning in youth or the early twenties. A history of other members of the family with the same trouble, also occurring in early life, is helpful in making a diagnosis. The hearing test; all tones reduced on an even scale and with increased bone conduction at first, lessening as the affection progresses. Of course, treatment by inflation is useless. Such a picture as this I have been accustomed to call otosclerosis. I realize that such a process may be existent and unrecognized by the overgrowth of the other type of deafness which I hope to describe. Before speaking in detail of the second type, in which I am greatly interested, may I mention the fact that in certain cases of decreased hearing due to a hypothyroid condition, the audiometer curve is very similar to that of otosclerosis and the two are not to be confused.

In 1921, Dr. F. P. Emerson, of Boston, delivered an address upon chronic progressive deafness before this Society. He believed that there is a gradual change in the organ of Corti, coincident with the pathological changes in the middle ear; that difficulty in hearing the whispered voice and a lowering of bone conduction for the middle and high tones are constant symptoms of this process. Since that time his theories have been substantiated by the general use of the audiometer and the pathological investigations and analyses of the audiograms by Dr. Stacey Guild, of Baltimore. Dr. Guild showed that the loss of the middle and high tones is due to atrophy of cells, the organ of Corti and ganglion cells. According to Dr. Emerson, there is a slow but constant toxic absorption which causes the cell atrophy, due to foci of infection. He advised the eradication of all such foci when possible and gave instances of improvement in hearing in certain cases. (Time does not permit me to give more than a resumé of the entire subject.) The symptoms of this class of patients are similar. They complain of a gradual loss of hearing, varying with the weather and with head colds; often tinnitus and a feeling of fullness in the ears. The physical findings are also similar, with only a difference in degree. There is more or less thickening and retraction of the drum membrane. Frequently the posterior segment is relaxed, due either to

autoinflation by faulty blowing of the nose, or to treatment by inflation. The mucous membrane of the nose and throat and nasopharynx is congested. The lower turbinates are usually enlarged. Further examination shows a small middle meatus, due to either a highly deviated or thickened septum. The tonsils, if present, are infected. The result of examination of younger persons is similar, with the addition of the presence of masses of lymphoid tissue about the Eustachian tubes and along the lateral walls of the pharynx. There is generally a history of carious teeth having been removed and often devitalized teeth still remaining. This short description of the physical findings may be constant or recurrent. The audiogram of the middle-aged adult shows a moderate loss of low tones and a sharp decline of the high tones. The bone conduction curve follows the air conduction curve. In younger patients the low tones may be normal, with a beginning decrease in the high tones. The causes of loss of hearing in these cases are: *1.* Mechanical obstructions to sound conduction; *2.* pathological changes in the internal ear, reducing sound perception. Now what are the most important factors in these conditions? Dr. Emerson very clearly explained his theories upon the subject, namely, infection of tonsils, accessory sinuses, etc. A very important cause of this train of symptoms is insufficient nasal breathing.

It is unnecessary to describe the anatomy of the nose and throat to this audience. You all know the importance of the middle meati, which is the common drainage area of the antra, frontal sinuses and ethmoid cells, and also the importance of the middle turbinate, where Nature has placed the olfactory nerve endings in just the best position to guard the individual against deleterious odors, a function of greatest importance in the dog, for instance. It is the surgery of this area which I hope to accentuate to you today. The nose is a most important organ; just realize what a wonderful air-conditioning apparatus it is. The air is filtered, moistened, and warmed before it is inspired; it flows over the anterior end of the lower turbinate and spreads through the middle meatus; the ostea of the accessory sinuses are in a position for the interchange of air; it then flows backwards around the Eustachian tubes and along the pharyngeal walls, and then is inspired. All mammals are nasal breathing, and man is the only one that can survive as a mouth breather. Nor-

mal nasal breathing depends upon a normal nose, and that is impossible unless it is used for breathing. Upon nasal breathing depends the development of the antra and a normal upper jaw and normal teeth, and upon the teeth depends the proper preparation of our food before ingestion, so you see what a responsibility a good nose has. The lack of normal breathing is caused by obstructions either in the nasopharynx, due to an abnormal growth of adenoid tissue, undeveloped anterior nares, or internal obstruction. The latter form is caused by hypertrophied lower turbinates or deviated septa. In my early years of practice it was the theory that both children and adults should sleep as much in the open as possible, irrespective of the temperature, season, or weather. Sleeping porches and balconies were in vogue and many people prided themselves upon the fact that water often froze in their bedrooms! A mental picture of the average child, prepared for its nap or a "good night's sleep" is always before me. The child flat upon its back, no pillow,—blankets, pinnned with huge safety pins, tightly fastened across its body, leaving a large ventilating shaft upon each side,—then the windows opened to the fullest extent. When I gently expostulated to the mother, I was informed that "Johnny must have a flat back" and also be "hardened." This was the usual habit in those days, practiced by all pediatricians and others. In my neighborhood, it was not until an experimental study of a series of children was made at the Children's Hospital that this pernicious practice gradually disappeared. Even now one meets with the health enthusiast who complains that he has increasing difficulty in breathing through the nose during the day and wonders why. He informs you with much pride that he opens the windows wide, irrespective of wind or weather, and that he often wakens with the puff over his head! Then I tell him that he has a little useful animal instinct left and should apply it. All this is a long story, but a common one. Mouth breathing is the natural result of overworked turbinates. They try to warm the excessively cold, damp air, and after a time they hypertrophy, so that even day nasal breathing is difficult. The recumbent position with passive congestion is an important causative factor.

Sir St. Clair Thomson, in his chapter upon deviated nasal septa, states that such malformations are more common in

"civilized" races, and he refers to the work of Catline, who found that they did not exist among the American Indians. Catline describes the Indian mother's custom of pressing the lips of the young baby together and keeping the mouth closed by bending the head so that the chin almost rests upon the sternum. He also comments upon the perfect condition of the teeth and the fact that overlapping teeth were unknown. There is no doubt that mouth breathing tends to the hypertrophy of both the adenoid tissue and the tonsils. James Love, of Glasgow, remarked years ago, "Show me a mouth breather and I'll show you a large adenoid growth." Our problem, then, is to prevent abnormal breathing in the young and to correct resulting deformities in the adult. I realize that the "pillow" of the American Indian would be difficult to make fashionable among the mothers of today. They can, however, be taught to use every endeavor to watch their children in this regard. I suggest a posture upon the stomach, with a very small pillow, or upon the side, with a large pillow. A bedroom, the best description of which is "a cool, fresh-smelling room"—with as few light-weight bed clothes as possible. Instead of adding additional blankets on especially cold nights, lessen the ventilation. Remember, "the body must breathe." Everyone knows the effect of cold air upon an inflamed congested nasopharynx—it is like adding an insult to an injury. To prevent chronic progressive deafness, we should do all in our power to insure normal nasal breathing under as natural conditions as possible by hygienic measures and surgical elimination of obstructions. Hypertrophied lower turbinates, especially with posterior ends, can easily be treated by electric coagulation. This must be done very cautiously, and not attempted if the hypertrophy is probably of adolescent origin. Generally the most important and most commonly seen obstruction is the deviated septum, blocking either one middle meatus or both. There is little air space and the middle turbinates are in close contact with the outer and septal walls. Occlusion may be due to a thickening of the cartilage at the junction with the perpendicular plate of the ethmoid bone or a marked bend of both structures. Frequently the middle turbinate is seen with difficulty, even after shrinking the mucous membrane. It is only natural to expect that with each and every head cold there is insufficient aeration of the accessory sinuses and difficult drainage, with a "hanging on" of the infection for weeks. Transillumination and X-ray

may show an acute condition which usually improves in time, but I doubt if the lining membrane ever returns to normal condition. I realize that it is often difficult to decide "just when to operate." The question of the growth of the nose, the danger of the tip of the nose "falling" from an injury, must be considered. But just the same, it is possible to remove the offending portion, even if a thorough operation is not done. The resection of the septum is performed either under local or general anesthesia. Inasmuch as it is always advisable to investigate the condition of the nasopharynx, general anesthesia is preferable. While it is well to remove basal spurs, ridges, etc., it is especially important that the mucous membrane and the perichondrium be deflected, so that the highly deviated portion may be removed, otherwise our object is not achieved. The flaps should be closely applied by means of suitable packs and removed in 12 to 24 hours. Perforations of the membrane are unfortunate, but if large, really do little harm. The operation should not be done in the presence of any unusual infection. If there is compensatory hypertrophy of either middle turbinate, the anterior end should be removed with a snare. In fact, if there is any doubt in one's mind regarding the free drainage of the ostea, a small portion should be removed to insure the success of the operation.

Our second area of interest is the nasopharynx. The absorptive rapidity of the nasal and pharyngeal mucous membrane is well known. We know that normal flora of bacteria in the nasopharynx must exist to preserve a normal balance of resistance and immunity. The difficulty is in preserving that balance. Proper nasal breathing will tend to prevent the excessive growth of pathogenic bacteria in the nasopharynx. The growth of lymphoid tissue about the Eustachian tubes and the walls of the throat, due to very cold air, becomes infected, maintaining an area of infection which has far-reaching and serious results, such as infection of the middle ears, either mild or serious, with a "neuritis of the organ of Corti," neuralgia, arthritis and symptoms of general septicemia. In removing masses of lymphoid tissue a general anesthetic is indicated. Direct inspection is necessary and can be obtained by elevating the soft palate. When a clear view is impossible, adhesions about the Eustachian tubes may be cut, or a portion punched out, by inserting the instrument

through a lower meatus and directing its position with the finger of the other hand. A simple cutting of adhesions does not suffice, a portion must be removed to prevent reformation. (In cases where the Eustachian eminence is absent, it is often possible to remove a small mass of lymphoid tissue from the fossa of Rosenmüller so that a slight prominence may be gained.) No one ever admits that he is a mouth breather, or that he snores, so it is very important that every attempt be made to keep the patient from sleeping upon his back after an operation. If he still persists in mouth breathing, then chin straps, bandages and adhesive tape may be tried for this purpose. In certain cases X-ray is helpful, but I believe surgical removal under direct vision is more satisfactory. In older patients, where the high tones are gone, the chances of improving the hearing are necessarily slight. I believe that the progress of the deafness can be arrested, and often one will be surprised to find that the hearing for the lower tones improves. Even if one does nothing but remove a small portion of the middle turbinate, the results are surprising; anything to give better drainage of accessory sinuses and lessening of nasopharyngeal congestion. Inflation and treatment of the Eustachian tube alone does not suffice—an attempt to remove the causes of the symptoms of deafness *must* be made. In older patients, where there is a senile atrophy, treatment of any kind is useless. Encouragement, attention to health, and possibly a hearing aid will make them more comfortable.

For several years I have been very much interested in a number of children who are deaf mutes. This class of cases presents a picture of an absolutely closed Eustachian tube. The tympanic membranes are intensely retracted and practically adherent to the promontory and inner tympanic wall. It is impossible to perceive any motion of the malleus with the otoscope and the membrane itself seems to be atrophic. In certain of these cases I have been able, under general anesthesia, by means of a catheter, to pass a bougie through the Eustachian tube, and in other instances this has been impossible, as there is apparently absolute occlusion. In the cases where I have been able to open by this method there has been an almost immediate evidence of hearing, and in three cases the children who were formerly mutes are now either talking normally or pronouncing words. It is my belief that

this condition is due to a lack of normal opening of the Eustachian tubes at birth. This experience leads one to suspect that there may be more cases of congenital occlusion of the Eustachian tube than one realizes. At least the subject warrants a more thorough examination of infants' ears at birth.

CONCLUSIONS.

1. Chronic progressive deafness is due to pathological changes in the middle ear, and coincident atrophic changes of the organ of Corti, etc.

2. The primary cause of this condition is deficient and abnormal nasal breathing.

3. Prevention and relief of chronic progressive deafness depends upon how early and how thoroughly obstructions to nasal breathing can be eliminated.

6 Bay State Road.

TUNING FORK TESTS REPORTED IN TERMS OF DECIBELS.*

DR. ROBERT J. HUNTER, Philadelphia.

Fifteen years ago Fletcher[1] proposed sensation units to measure the ratio of change in intensity of sound. Since then, decibels, as they were named in honor of Alexander Graham Bell, have been adopted as the standard in this and many other countries. They are also used to express the ratio of change in power level in acoustical instruments.

There is a great deal of discussion in the literature about the relative value of measurement of hearing impairment with audiometers as compared with tuning forks. Much has been written about the necessity of interpreting tuning fork tests in terms of intensity rather than in terms of distance or time difference of the patient's hearing compared with the normal observer. If the otologist had an easy method for attempting to measure the intensity of a tuning fork, his attention would be called to the varying factors which change the intensity of a fork and the advantage of an instrument like the audiometer would be apparent. The Committee on Hearing, of the Royal Society of Medicine, has suggested such a method.[2] They propose as a measure of the degree of hearing impairment the period of time in which a tuning fork decays to one-half its initial intensity. As a result of a study of their report, I have drawn up rules which can readily be followed by otologists without a knowledge of logarithm. I recommend this report of the Royal Society with one reservation. In it, through an error, two standards have been described as identical: one for the period of time in which the amplitude of a tuning fork decays to one-half its initial amplitude, during which change of amplitude the fork will decay 6 dcb.; the other for the period of time in which a fork decays to one-half the original intensity, during which change the fork will decay 3 dcb. It is the latter unit which we shall discuss in this paper, under the title of half-intensity unit. One great advantage of this unit is that it makes it possible

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for a doctor to measure his own forks. The method proposed depends on the fact that intensity of sound varies inversely as the square of the distance from the source. The zero or receptive point of hearing was said by Lord Rayleigh to be one-half inch inside the auditory canal. A fork is held $11\frac{1}{2}$ inches from the tragus, or 12 inches from the zero point. When it can no longer be heard, a stop watch is started and the fork moved to one-fourth inch from the tragus, or three-fourths inch from the zero point. When it can no longer be heard, the watch is stopped. Note that three-fourths inch doubled four times equals 12. Each time the distance is doubled the intensity becomes one-fourth and there are two half-intensity periods; therefore, divide the time noted with the watch by eight to get the half-intensity period of the fork. Of course, other distances, that are even multiples of each other, can be used. If we hold the fork first at 12 inches and then at three inches from the zero point, we divide the time difference by four. A method for calculating the half-intensity period of a fork for bone conduction is also described. This method makes it possible to measure the change in the half-intensity period, depending on the change in decrement of the fork, which will be caused by pressure on the skull. The British method simply proposes the half-intensity period of time as a constant for the fork, instead of the decay per second.

Let us now see the relation between the half-intensity period of a fork, the decrement of a fork to the base ϵ and the decibel decay per second of a fork. Ordinarily, a decibel is said to be the smallest appreciable increase in the intensity of sound that the human ear can detect, although a laboratory worker can detect an increase of 0.4 decibels.[3] It expresses the ratio of change of intensity level in terms of common logarithm.

The mathematical formula for measuring decibels is

$$10 \log_{10} \frac{I}{I_0} = \text{dcb.}$$

and the formula for measuring tuning fork intensity is

$$\frac{I}{I_0} = \epsilon^{-kt}$$

Therefore, bels are the exponent of 10, the base of common logarithm and the decrement "k" is the exponent of ϵ

(2.71828), the base of natural logarithm; therefore, if we multiply the decrement by the modulus we convert it into bels and it follows that if forks have been standardized in the usual way, the decrement multiplied by 4.34 will equal the decibel decay per second. This has been obvious to mathematicians but has not been emphasized by otologists.

It is proved as follows:

$$\text{Taking } 10 \log_{10} \text{ of the formula } \frac{I}{I_0} = \epsilon^{-kt}$$

$$\text{we have } 10 \log_{10} \frac{I}{I_0} = 10 \log_{10} \epsilon^{-kt}$$

$$\text{decibels} = -10 \text{ kt } \log_{10} \epsilon = -4.34 \text{ kt} = \Delta \text{ t}$$

$\therefore \Delta = -4.34$ k, the intensity logarithmic decrement and the decibel decay per second of a fork.

The total loss is obtained by multiplying the decrement by the number of seconds the fork can be heard by the normal examiner after the patient has ceased to hear it. We find that Fletcher,[4] in his paper before the American Otological Society in 1925, recommended the very simple and direct method of having the forks calibrated in decibel decay per second. In a recent report before this Society, it was advised that this should be done by comparing the intensity of the fork with a well standardized audiometer and noting the time required to decay until the sound of the fork becomes inaudible.[5] The distance the fork is held from the ear must not vary while the decay is being timed with the stop watch. When using the half-intensity method we divide the time difference of hearing by the number of seconds in a half-intensity unit. The number of units multiplied by three will give the total loss in decibels. That the unit is equal to 3 dcb. can be shown as follows: Let the intensity "I" be one-half the intensity "I_0"

$$\text{then } -10 \log_{10} \frac{1/2}{1/1} = 10 \log_{10} \frac{1/1}{1/2} = 10 \log_{10} 2 = 3.0103 \text{ decibels}$$

which can be more graphically shown as follows: Let 10 be a given intensity and let it be raised in steps each about 25 per cent more than the preceding one. Each of these will be about 1 dcb. Then we have

	10	12.5	—16	20
and again				
	20	25	—32	40

It will be noted that each time we go down three steps the intensity is halved.[6] That each step is about 1 dcb. is shown

by the fact that the logarithm of $\frac{I}{I_0}$ must be [.1] when the equation equals 1 dcb. The nearest mantissa is .1004, which corresponds to the number, 1,259, from which we subtract unity, 1,000, to get about 25.9 per cent increase of energy level with each decibel.

Another point about the question of half-intensity time which has not been emphasized is a relation to $\log_\epsilon 2$ which affords a very simple way of finding either the decrement or the half-intensity time if one of these factors is known.

Take the formula quoted above:

$$\frac{I}{I_0} = \epsilon^{-kt}$$

Let $$\frac{I}{I_0} = \frac{1}{2}$$

Then $$\frac{1}{2} = \frac{1}{\epsilon^{kt}}$$

and $$\text{Log}_\epsilon 2 = kt$$

Therefore, $\frac{.6931}{t}$ = the decrement when "t" is the time in seconds that it takes a tuning fork to decay to one-half the intensity it had when the measurement began.

We are now in a position to solve some other simple problems which are dependent upon the facts already demonstrated. We have seen that a half-intensity unit is equal to $10 \log_{10} 2$, which is 3.0103; therefore, if we know the decibel decay per second of a fork, we divide 3.0103 to get the number of seconds in a half-intensity unit for that fork. We have already demonstrated that in a similar fashion, decrement (to the base ϵ) divided into $\log_\epsilon 2$, which is .693147, gives the number of seconds in a half-intensity unit. Knowing the number of seconds in a half-intensity unit, we can readily obtain the decay per second either to base ϵ (decrement "k") or base 10 (bels per sec.) by dividing the appropriate log 2 (*viz.*, .6931 or .30103). Given a decibel loss, we divide by three to get the number of half-intensity units a fork should give for the same loss. Each time the distance from the source

of sound is doubled or halved, the intensity changes 6 dcb. When reading foreign articles we must remember that there are two international units for measuring intensity of sound. These units are the bell and the neper (named in honor of Bell and Napier). The formula for the latter is

$$1/2 \text{ Log}_{\epsilon} \frac{I}{I_o} = \text{nepers}$$

To change nepers into decibels, multiply nepers by 8.68; 1 dcb. equals 1.151 decinepers.

The inquiry is frequently made: Is a decibel an amount, like a peck of potatoes? We have stated that a bel is the exponent of 10, representing the energy required to raise a given sound from one point to another; therefore, a change of 10 dcb. will not always indicate the same amount of energy. The energy required to raise the decibel level can be expressed by adding to one, as many ciphers as there are bels. For instance, if we raise a sound 10 dcb.; that is, from 80 to 90 dcb., the energy difference is the difference between one followed by nine ciphers (1,000,000,000), and one followed by eight ciphers (100,000,000); whereas, if we raise the intensity 10 dcb., that is, from 20 dcb. to 30 dcb., the difference in energy is expressed by the difference between 1,000 and 100.

For office use, it is an advantage to have the forks calibrated in both half-intensity units and decibel decay per second, because either one factor or the other may be more easily used for rapid calculation. Sometimes the fractions can be changed to the nearest tenth without causing a significant error in 100 dcb.

It would be misleading to leave the subject of interpreting tuning forks in terms of decibels without calling attention to the well known difficulties met in measuring the decrement of a fork. The decrement has been defined as "the time rate of decrease of the logarithm of the loudness,"[2] or "the logarithmic decrement of the oscillations."[7] Each vibration of the prongs is said to be a little less than the preceding one, going down in geometric progression in a fashion the reverse of compound interest, but these are theoretical observations. Davis[8] states that the rate of decay is greater for large amplitudes than for small. The necessity for forks of high quality is recognized in the literature and by the manufacturers of

forks. Such forks, carefully standardized by a physicist and used with meticulous care, may give fairly uniform results. The best sets cost from $150 to $400. In the few tests that I made with the best grade of forks (Bezold-Edelman), calibrated by ear, the difference between the decibel loss obtained with them and the decibel loss obtained with ordinary forks was not significant. Aside from the qualities of a fork for accuracy of pitch, volume and regularity of decrement, the manner in which a fork is held in the hand will affect the decrement and, therefore, the intensity. This fact has led to innumerable directions in regard to the manner in which the fork is to be struck, the part of the stem that is to be taken hold of, and the firmness with which it is to be held. All of these variants make it difficult to test accurately with tuning forks, and even if a doctor acquires a "proper" technique, it is difficult or impossible for others to copy it exactly. In an attempt to discover what relation, if any, would be found between the decrement as measured by ear and the decrement as measured by physical means, I have had some forks examined by various methods. For this purpose I selected ordinary forks of the kind usually found in office practice. I found that attempts to measure the amplitude required that the fork be held in a holder, which at once changed the decrement from what it would be when the patient was examined. The method of reflecting the vibrations on a scale was tried. The blow required caused a very large amplitude with large decrement at the beginning, which soon lessened a great deal. Any attempt to wait until this subsided made the period for measurement too short. Photographing the vibrations was tried. Some forks were sent to a commercial laboratory. The exact method used was not stated. It was "a visual method of measuring the rate of decay with the fork held in the hand in the same way as held when making medical tests." This method was not found practical for two of the forks in which "the rate of decay for large amplitudes was more than twice the average rate for the whole audible period. For this reason the audible times for these two forks were measured in a quiet room and the decay was calculated, using the audible time of a known fork as standard." I measured some forks in a soundproof room with a noise level apparatus. The change of decrement was not so great, because it was not necessary to strike as violent a blow as with the other methods. With the higher forks, the decay was so rapid that only a few read-

ings could be made. With two forks with smaller decrements, it was possible to check the time at 5 dcb. intervals. When plotted to get the rate of decay, a 256 fork decayed 4, 3½, 3½, 3, 3 and 3 dcb. at 10-second intervals. A 171 fork decayed 7, 6, 6 and 5 dcb. at 20-second intervals. The forks were held in a split cork in a laboratory stand and also suspended on a string. The decrement was not the same when the fork was held tightly as when it was held loosely or when it was vibrating freely, suspended on a string through the crotch. When the fork was held in the hand one-fourth inch from the microphone of a noise level apparatus* in a soundproof room, although the hand showed no perceptible tremor, and was apparently held in exactly the same position, yet the oscillations on the dial of the metre were too great to read. If the fork was held further away from the microphone, say three inches, the instrument was not delicate enough to register the sound for a sufficient period to make a calculation. The forks used did not have sufficient volume. I then measured the decrement by ear by the method described, holding some of the forks three-fourths of an inch from the zero point of hearing and some three inches from the zero point. With this method, changes of decrement due to various factors came into play only during the interval of time that the fork was heard at the near point after it had ceased to be heard at the distant point. As the fork died away, the decrement was more constant. A new set of conditions now made the measurements variable. It was difficult to hold the fork still. The accuracy of the measurement of the distance from the ear, attention, acuity of hearing, fatigue and varying noise level of the room had to be considered. We can see that if the noise level of the room varied just as the fork reached the minimum frequency for hearing, the readings would vary; therefore, in calibrating by ear, an alert person must be tested in a quiet place. The average of a number of tests on many people is to be preferred. Thus, we shall see that the decrement of a fork measured at a physical laboratory will not be the decrement found when examining a patient.[9] The following chart shows decrement calculated by several methods:

DECREMENT OF TUNING FORKS IN DECIBELS PER SECOND.

Measured with an Electrical Resistance Products Corp. noise level metre in a soundproof room, by ear in an ordinary room, by a commercial laboratory and by a physicist. The forks were held before the microphone on an iron stand:

*Electrical Resistance Production, Inc., Type R. A. — 142a.

(a) Tightly held in a cork, measured in a soundproof room.

(b) Lightly held in a cork, measured in a soundproof room.

(c) Suspended on a string through the crotch, measured in a soundproof room.

Also

(d) Held in the hand, ordinary room, measured by ear, ¾″ x 12″.

(e) Held in the hand, ordinary room, measured by ear, 3″ x 12″.

(f) Measured at commercial laboratory.

(g) Measured at physics laboratory, condenser, amplifier, camera, fastened to stand.

(h) Measured at physics laboratory, condenser, amplifier, camera, held in hand.

Pitch	Tightly	Lightly	Suspended	In the Hand by Ear		Commercial	Physics Lab. On Stand	Physics Lab. In Hand
Steel—	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
c_1 256	0.977		1.110	0.2768	0.381			
c_2 512	0.555	1.179	0.769	0.4280	0.320			
c_3 1,024	5.900	6.500	1.560	0.8850	1.003			
c_4 2,048	2.739	1.652	1.538	1.54	1.075			
Magnesium—								
c_5 4.096	3.420	4.556	4.440	4.000				
c_1 256	1.38	0.862	0.5464	0.255				
c_2 512	1.28	0.7934	0.625	0.382	0.351	0.918		
Steel—								
171	0.296	0.28	0.289	0.116		0.620	0.640	0.390
A 435	0.784	0.833	0.892	0.316		0.9		
A 435 N	0.75	0.65		0.257		0.79		

Note: The figures in column (a) suggest a technical error. It may be due to vibration of the iron stand in which the forks were firmly clamped. Columns (b), (c) and (d) are successively smaller, as a rule.

The A 435 forks are identical in appearance and of the same make. They are used for the Stenger test. Note that the decrement is not the same. When given identical blows, one fork stops vibrating before the other. This fact must be considered in making the Stenger test.

A patient was examined with the audiometer and immediately afterward with Hartmann forks and with Bezold-Edelman forks, using the calibration obtained by ear for the latter to see how it compared with ordinary forks. This is shown in the following chart:

	Right Ear			Left Ear		
	C_1 256	C_2 512	C_3 1,024	C_1 256	C_2 512	C_3 1,024
	Decibel Loss of Hearing					
MacFarlan audiometer	50	50	45	50	50	45
Hartmann fork calibrated by noise level apparatus while suspended	68	32	45	69	30	45
Bezold Edelman fork calibrated by ear; distance, ¾″ x 12″	24	21	17	21	21	19
Hartmann fork calibrated by ear, 3″ x 12″	23	18	30	24	17	30
Bezold-Edelman fork calibrated by ear, 3″ x 12″	17	17	16	15	17	17

We can see from the table that the decibel loss found with a fork will depend entirely on which figure is picked as the decrement. If the object is to make the fork reading correspond with the audiometer reading, the method of calibrating by matching the loudness of the fork with the intensity shown on an audiometer should be very satisfactory. I have not tried this method. If no audiometer is available the method of testing by ear gives results that are fairly uniform for the fork concerned, and if forks must be used I would advise that the records be kept in decibel loss so computed. If the fork is frequently calibrated, attention will be called to the variation in the figures obtained. The mean of many tests will, of course, give an average reading for the fork and technique used.

The appointment of the Committee on Standardization of Audiometers, of the American Medical Association[10] and of the American Standards Association,[11] indicates that the need of maintaining a standard for audiometers is recognized. A plus or minus variation of 5 dcb. is permitted, which is equal to a spread of 15 dcb. This is equivalent to five half-intensity units of a fork, which, according to the above standard, would permit that much variation with different tuning fork techniques. Dependable audiometers are on the market. They will, no doubt, be improved, but tuning forks will always vary; therefore, the rules for interpreting tuning fork tests are given in this paper with the hope of improving the method of examination with forks, but not with the idea that they can take the place of audiometers. In an effort to see how the decibel loss determined by tuning forks compared with the loss shown with audiometers, two alert, intelligent patients with conduction deafness were tested with four audiometers

of different makes and immediately afterward with forks. The decibel loss shown by the forks was calculated, using the decrement obtained by the noise level apparatus and also the decrement obtained by ear. The results were so varied that no conclusions could be drawn, except the fact that when the decrement found by ear was used, the decibel loss averaged from one-third to one-half the loss shown by the audiometers. The decrement obtained by physical means gave sometimes lower and sometimes higher loss than the audiometer reading.

CONCLUSION.

The manner in which tuning fork intensity can be interpreted in terms of decibels is discussed and attention called to the significance of the numbers, 4.34, .6931 and 3.0103. The decrement of tuning forks varies so much under different conditions that a very careful technique would be necessary to get uniform results for a given observer. The technique could not well be copied by another observer; therefore, the conclusion is drawn that audiometers are the most dependable instrument in measuring the intensity of impairment of hearing.

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DISCUSSION.

DR. HARVEY FLETCHER (New York): I am sure that it is timely to go over this subject again. I was very much interested in Dr. Hunter's paper; however, I think the picture of tuning fork behavior presented in his paper is more pessimistic than it need be. I believe that the type of tuning fork he used in his tests was greatly affected by the different ways in which it was held or mounted, and that this influenced the results and his conclusions.

I would like to take a few minutes to discuss a method of expressing the decrement of a tuning fork.

(Slide) When a tuning fork vibrates, you cannot see it vibrate, but if the vibration were magnified a large amount and I pulled up this screen while the fork was vibrating, it would make a trace like that. It is evident that the amplitude of the vibration decreases as time goes on.

If the initial amplitude is represented by A_0, the amplitude after one complete cycle by A_1, the next amplitude by A_2, the next by A_3 and so on, it has been found that the ratio of each amplitude to the amplitude immediately preceding is a constant mathematically expressed

$$\frac{A_1}{A_0} = \frac{A_2}{A_1} = \frac{A_3}{A_2} = \frac{A_4}{A_3} = \bar{O}$$

where the constant "$\bar{O}$" is called the decrement. It is the fractional decrease in amplitude for each complete cycle of vibration. The logarithmic decrement "k," to which Dr. Hunter referred, is defined as

$$k = \log_\epsilon \bar{O}$$

If the number of complete cycles or vibrations executed by the fork per second is "n," then the fractional reduction in amplitude per second is

$$\frac{A_n}{A_0} = \bar{O}^n.$$

If the fork vibrates for "t" seconds then the amplitude reduction is

$$\frac{A_{nt}}{A_0} = \bar{O}^{nt}$$

Since the intensity of the sound emitted by a fork is proportional to the square of the amplitude

$$\frac{I_{nt}}{I_0} = \frac{(A_{nt})^2}{(A_0)^2} = \bar{O}^{2nt}$$

But by definition the decibels reduction "a" in the time "t" is given by

$$a = 10 \log_{10} \frac{I_{nt}}{I_0} = 20 \text{ nt } \log_{10} \bar{O}$$

$$= 20 \text{ nt } \log_\epsilon \bar{O} \log_{10}\epsilon = 20 \text{ nt x } .434 \text{ k}$$

This shows that the decibels reduction in the intensity of the sound given off by the fork is proportional to the time in seconds, or

$$a = \Delta t,$$

where the constant Δ is related to the logarithmic decrement "k" and the frequency of the fork "n" by

$$\Delta = 8.68\ nk$$

Since Δ is negative, "k" is also negative. For example, in one fork on which measurements have been made, Dr. Fowler's 512 fork n = 512, Δ = 1.31, k = —.000295, and $\bar{O}$ = .9997. The fork vibrates 512 cycles per second. The sound given out by it dies down at the rate of 1.31 dcb. per second. The actual decrement "O" in the amplitude is .9997, or the amplitude is only reduced .03 per cent on each swing. The reduction in amplitude per second is "$\bar{O}^n$," or

$$(.9997)^{512} = .86$$

In 10 seconds the amplitude is reduced to .22 of its initial amplitude. In 20 seconds it is reduced to .22 x .22, or .05; or 5 per cent of its initial amplitude, or 26 dcb.

At an international conference last summer it was agreed that decrements should be expressed in terms of decibels. I made a plea to the Physical Society last week, that they talk of decrements only in terms of decibels. I hope that the otologists may likewise accept this method of expressing decrements, and that they will have their tuning forks calibrated in decibels per second. Then, having determined the time in seconds that the average normal ear can hear the fork, the hearing loss of any patient can be determined in decibels by subtracting the time in seconds during which he heard the fork from the normal hearing time and multiplying this figure by the constant of the fork expressed, as suggested above, in decibels per second.

THE USE OF THRESHOLD AND LOUDER SOUNDS IN CLINICAL DIAGNOSIS AND THE PRESCRIBING OF HEARING AIDS. NEW METHODS FOR ACCURATELY DETERMINING THE THRESHOLD FOR BONE CONDUCTION AND FOR MEASURING TINNITUS AND ITS EFFECTS ON OBSTRUCTIVE AND NEURAL DEAFNESS.*

DR. EDMUND PRINCE FOWLER, New York.

There appears to be much confusion as to just what may be accomplished by the use of audiometers and hearing aids and what standards should be used in judging these instruments.

Almost daily someone asks me as to the relative merits of this or that audiometer, and this or that hearing aid. I reply that any instrument which meets and maintains by efficient service the minimum standards set up by the Committee on Physical Therapy of the American Medical Association should be satisfactory if marketed by a financially and ethically reliable firm. Avoid complicated and tricky machines and charting methods. They are ill-advised and confusing, and often serve to cover up defects in calibration of frequencies and intensities. The graphs are often meaningless and cannot be compared with standard audiograms. Many such questionable machines have been foisted upon the schools in New York and other States. They tend to discredit audiometry. The basic requirements in machines and charting were set forth before this Society by Mr. Wegel and myself in 1922.†

During the past 17 years several new phenomena have been observed, which, step by step, have advanced our knowledge of the physiology of hearing, the testing of hearing, and the prescribing of hearing aids. One of these, the "recruitment of loudness with intensity," enables one to diagnose obstructive from nerve deafness without the use of bone conduction, and possibly to differentiate some of the lesions occurring

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†Trans. Amer. Otol. Soc., and those of the Amer. Laryngol., Rhinol. and Otol. Soc., 1922.

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with both of these types of deafness. A not inconsiderable virtue of audiometric experience has been the exposure of many defects inherent in or imposed upon the older and newer testing methods. Without accuracy in measurement it would have been impossible to discover the newer phenomena or to obtain dependable interpretations for their clinical use. Many of the observations formerly reported bear repetition because they still generally hold true. They may be reproduced only by properly calibrated audiometers.

FUNDAMENTAL OBSERVATIONS.

1. From the standpoint of audiometry, the logarithmic scale is logical.

2. The logarithmic frequency scale in which equal intervals on the scale correspond to equal logarithmic intervals of frequency is the more logical to use in an audiogram. This is the musical scale.

3. a. The intensity scale in which the energy or sound pressure is used, and *b.* that in which the sensation is given as proportional to the logarithm of the stimulus are both available, but hearing curves plotted on these two scales will, in general, bear little resemblance to one another. The sensation scale is the more satisfactory for most purposes.

4. No ear has an absolutely smooth hearing curve. Even the normal ear has undulations which deviate up and down at different frequencies.

5. Although the variations in audiograms are considerable, the curves of hearing of the two ears of one subject are very often strikingly similar in certain respects (the patterns are similar). This is so in spite of the fact that there are fully as many different ways in which a given lesion may affect an ear as there are anatomical and physiological variations in normal ears.

6. It was thought that the low point of intersection of the two normal curves of threshold audibility and threshold feeling (touch) could be defined as the lower tone limit. This was estimated to be at about 20 d.v. The upper tone limit was similarly extrapolated somewhere between 20,000 and 25,000 d.v. More recent determinations make it questionable whether any such definite limits can be determined. The older tuning fork measurements defining these limits were faulty because of neglect to measure the effect of intensity.

7. Readings taken at frequency intervals of not over one-half to one octave give sufficient accuracy for present clinical purposes.

8. The tone from an oscillator and receiver should be so pure that harmonics cause less inaccuracies than the observational errors.

9. Charts showing the effects from cotton, wax and vaseline ear plugs in the external meatus indicate no simple law of sound transmission.

10. An audiogram chart using a straight line to represent the average normal curve for both air conduction and bone conduction was advocated. This greatly simplifies the picture and avoids the old and persistent fallacy of stating that bone conduction is better or worse than air conduction, depending on the absence or presence of nerve deafness. Bone conduction is seldom, if ever, less than air conduction, in nerve-deafened ears or in any other ears. It sometimes appears to be less because of the confusion in statement and interpretation of the data elicited. Bone conduction is never more than 10 to 15 dcb. below air conduction if data are correctly determined. This is within the normal variations often occurring from moment to moment without discoverable cause. Great care is necessary to distinguish between hearing and feeling by bone conduction for frequencies below 1,000. A method for detecting false bone conduction determinations will be described anon. A dependable diagnosis is impossible unless both air conduction and bone conduction are taken at a spread of frequencies; 256, 1,024 and 4,096 have proved satisfactory for minimum preliminary audiograms.

11. Errors, normal or skew, in observation and sampling were pointed out; the skew errors being those that occur asymmetrically above and below the average.

12. Attention has been repeatedly called to the fact that *a.* loss of high tones occurs not only in nerve deafness, but also with certain obstructive lesions (see Fig. 1); *b.* loss of low tones occurs not only in obstructive deafness, but also with certain neural lesions; *c.* both the high and low tones may be heard normally by air conduction and bone conduction with marked loss of hearing in the middle tones (for air conduction and bone conduction) (slide XYZ).

13. The possibilities and limitations of the audiogram as a basis for prescribing hearing aids were discussed.

14. Case reports with audiograms showing the effects of various impedance and neural lesions (precipitous drops wholly in the upper register, the shifting of the brow of the drop with progressive deafness, and dips, loops and so-called tone gaps were demonstrated.

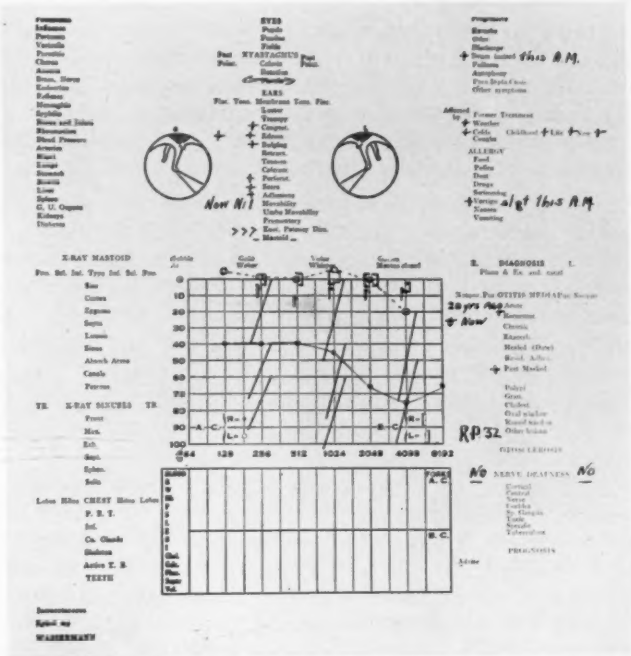

Fig. 1. Notice that both the normal (masked) bone conduction and the unchanging slopes of the loudness balance lines indicate no extensive neural deafness in the right ear.

15. The importance of masking the opposite ear in bone conduction tests and always in air conduction tests if the threshold is down over 45 dcb. for the middle frequencies.

THRESHOLD AND LOUDER SOUNDS.

From time immemorial, it has been the custom (because of necessity) to estimate hearing acuity by the ability to hear the faintest audible sounds; in other words, the hearing has

been measured at minimium audibility. In this way, the audiometer, the tuning fork, voice, acumeter, watch's tick, etc., were and still are usefully employed.

The ear, however, does not usually bring to consciousness minimum audible sounds. One does not, except under special circumstances and with special effort, listen to them; articulate speech (conversation) is not understood, and the slightest noise masks them out.

In order to obtain dependable threshold measurements, one must test in a soundproof room (in absolute quiet). This is obtainable only in a few research laboratories, but any carefully noiseproofed room is usually sufficiently protected for clinical testing, especially if the patient has a loss of hearing sufficient to shut out the masking effects of outside noise.

It is not necessary, or at times desirable, to test in a noiseproofed room for the purpose of prescribing hearing aids, because most patient's desire above all else to hear conversation clearly, and in the world in which we live, conversation is rarely heard except in fairly noisy surroundings.

The environment is usually quite different from a quiet testing room, and it is not, therefore, strange that our tests at minimum audibility should not give a complete picture of the ability to understand articulate sound at ordinary speech intensities; *i.e.*, 20 to 60 dcb. above normal threshold, depending on distance. In other words, if the hearing happens to be down on the average over 40 dcb. for the frequencies most used for speech, it means that the intensity of nearby unamplified voice sounds will be heard at not over 20 dcb. above threshold; that only about 50 per cent of the articulation will be clearly heard and understood. A greater loss of hearing, whether from ear lesions or masking noise, will permit little, if anything, to be clearly heard. The latter is well illustrated by the inability to hear clearly without amplification an ordinary voice from the stage of a large theatre, when one is in the back row of seats. The slightest nearby noise will mask out the actors' speech. Of course, many deafened people learn to get along with low volume reception, and normal hearing people can at times get along in the back row of the theatre, but everyone will testify to the strain involved.

Until the advent of the thermionic valve audiometer, it has been the custom (because of necessity) to estimate the hear-

ing acuity for very high frequencies by the ability to hear them or not to hear them. In this way the monochord and whistles were and still are used. If heard, it has been the custom to believe there was no loss, and that, therefore, the high tone levels were intact. This is, of course, not true. No quantitative estimation of the hearing for these tones can be thus obtained. Moreover, the high tones, being subject to the recruitment phenomenon, may show high thresholds, and yet a sound when loud may appear as loud as in a normal ear.

TIMBRE.

A definite pure tone or other sound may sound quite differently to different people, even though a careful examination reveals no ear abnormality. One's own voice may at times appear muffled and changed to one's self, and yet others may not notice any change in quality.

It seems to me that this, at least in part, is brought about by anatomical variations. No two cochleae are identical in bony or soft tissue dimensions. Variations in size of the cranial tissues and air (resonance) cavities alone cause a difference in the timbre of many sounds. Small mastoid cells act like filters if open to the middle-ear cavity, and so affect its resonance. This is certainly true for loud sounds, and may be demonstrated by applying a sound to one ear of several subjects, and listening to it through rubber tubing connecting the subject's opposite ear to the examiner's ears by a binaural stethoscope. Sounds heard in this way, both by air and by bone conduction (particularly the latter), certainly have a different quality in different individuals, and it would appear that any given individual may, therefore, receive a different sound pattern from anyone else. Moreover, even if the physical sound patterns in different cochleae be identical, the higher centres and cortices in different people will not be identical and will not function exactly alike; and, therefore, even identical nerve impulses will set up variables in quantity and quality of sensation. In other words, sensitivity, analysis and tone pictures in different brains will be different. No one knows exactly how a given sound appears to others. Word deafness (congenital or acquired), with apparently normal external, middle and internal ears, may in some instances be an exaggerated example. The remarkable thing is that, in spite of all these variables, it

is possible to determine a fair average normal hearing level for the human ear.

No matter what frequency or band of frequencies is missing from loud sounds, the healthy ear appears able to re-establish the missing frequencies. The diseased ear also does this to a greater or lesser extent, depending on the type and extent of the lesions.

It would appear that the neural mechanism is able not only to put back the missing frequencies, but to restore sensations of loudness. Limited lesions, those showing narrow gaps in the curve — even a loss of 70 or more dcb. in the narrow gap area — may, because of the pick-up from both sides of such a lesion, show near normal loudness for very loud sounds in the tone area in question. In other words, a total loss of neural elements may not show a total loss of hearing from the gap area.

TESTING AT THRESHOLD.

When testing at any frequency, if the threshold is approached from above or below, there is noticeable a difference in the certainty of the observations as between nerve and impedance (obstructive) deafness. The ear with uncomplicated, so-called nerve deafness responds with greater certainty. In consequence, the threshold is definite. It does not vary from moment to moment. It pops out suddenly from complete silence or disappears suddenly from definite audibility. There is little or no doubt in the mind of the listener. The sound is either heard or it is not heard.

On the other hand, the ear with uncomplicated impedance lesions (so-called conduction or obstructive deafness) responds with uncertainty to very faint sounds, and the plotting of the threshold level may vary greatly from moment to moment. The sound appears and disappears unobtrusively; it seems to have been there or to have been absent some time before the change is definitely realized. There is a lag in picking it up and in losing it. There is doubt as to whether it is heard or not heard. The examiner is forced to choose the minimum intensity which is obtained more often than the others, and plot this as the true threshold at each frequency. The plotting of such thresholds when using tuning forks is very uncertain, because of the constantly diminishing intensity.

This difference in ease of detection of threshold sounds is largely occasioned by the presence or absence of what I have called the "recruitment phenomenon," which is present in every hearing ear with "nerve" deafness. The recruitment phenomenon is usually demonstrated by throwing alternately into the two ears increasing intensities and balancing the loudness binaurally.* This I have called the method of "alternate binaural loudness balance." Its clinical application is increasingly valuable as new uses are discovered.

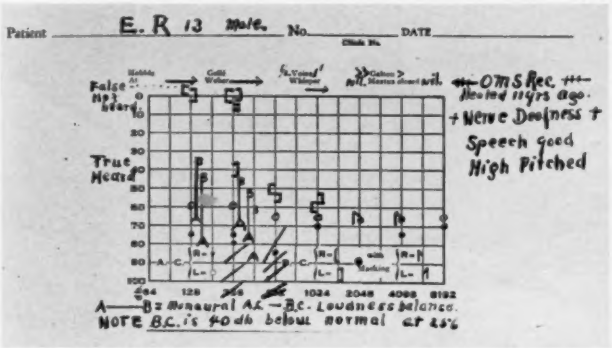

Fig. 2. The points at the extremities of the vertical lines joining A and B indicate the number of decibels above threshold necessary to balance in loudness air conduction with bone conduction, respectively. At 128 d.v. it is 30 dcb. At 256 d.v. it is 20 to 25 dcb. Notice that there is no change in the number of decibels between A and B as the applied sounds are increased in intensity.

TO TEST THE ACCURACY OF BONE CONDUCTION DETERMINATIONS.

Bone conduction measurements are often misleading, largely because of an inability to differentiate between hearing and feeling for the frequencies near and below 500, and because of crossed audition. Fig. 2 shows audiograms of two ears, both of which are markedly down by air conduction for all frequencies, and bone conduction for all above 256 d.v. For the lower frequencies, bone conduction appears to be near normal. If this latter is true, it is the custom to say that the neural mechanism is intact for the lower tones.

In the absence of nerve deafness or tinnitus, air conduction will approximately balance with bone conduction binaurally with equal increments of loudness above thresholds. Their

*Trans. Amer. Otol. Soc., 1936-37.

responses will be linear. Also, monaurally the ratio in the increment of loudness with increasing intensities will remain the same for both air conduction and bone conduction. In other words, at thresholds and at given levels above the two thresholds, sounds heard by air conduction will sound approximately as loud as the same sounds heard by bone conduction.

In this instance such is not the case, because for the left ear at 256 d.v. it required 40 dcb., and at 128 d.v., 35 dcb. above bone conduction threshold to balance in loudness 5 dcb. above air conduction threshold. With a normal bone conduction, there is no loss or gain in the ratio of loudness increase with intensity. Bone conduction, then, is not normal, but 40 to 30 dcb. below normal, at 256 and 128, respectively, and it may be even lower, because the sensation of feeling is always added to the sensation of hearing when using any low frequency. In this patient there is, then, indicated a nerve deafness affecting the low tones as well as the high tones. There is also some impedance (obstructive) deafness, as indicated by the spread between air conduction and bone conduction plotting, and by a history of recurrent suppurative otitis media, now healed. Of course, impedance (obstructive) lesions in the middle ear may, in some instances, account in whole or in part for a near normal bone conduction in the presence of 10 to 20 dcb. of nerve deafness, but when such is the case, there is no loss of loudness when using sounds well above the indicated normal bone conduction threshold. There would be, if anything, a recruitment, an increase in loudness.*

MEASURING TINNITUS AND ITS VARYING EFFECTS ON THE HEARING.

The effect of tinnitus on hearing acuity may be measured by the use of the "loudness balance" technique. In this connection it should be remembered that, although a high pitched tone has little or no masking effect on tones below it in frequency, this does not necessarily mean that a high pitched tinnitus, and the lesions causing it, may not affect the sensation of loudness for these sounds. Clinically, a diminution of tinnitus is often accompanied by an increase in hearing for the frequencies above, at and below that of the tinnitus (Slide H. O. P.). *Many medical and surgical procedures credited*

*The bone conduction was used here as a norm. It proved to be a subnorm.

with the cure or relief of deafness really act in whole or in part by their beneficial effect on the tinnitus.

To measure the loudness of a tinnitus, match the loudness of the tinnitus in one ear against the loudness of a similar frequency (or frequencies) put into the opposite normal or abnormal, nontinnitus ear. Tinnitus is not a pure tone. It is always made up of a band of frequencies. The number of decibels above the threshold of the nontinnitus ear required for a sound to balance the tinnitus in loudness, may be considered a measure of the loudness of the tinnitus as it appears to the patient. It will at times also be a measure of the contra- and ipsolateral masking possibilities of the tinnitus as such, and, therefore, of its effect, or the effect of the lesions causing it, on hearing acuity at the masking frequencies. It will make possible measurements of changes in the intensity of tinnitus.

It is not possible, monaurally, to balance tinnitus with like or unlike applied frequencies of varying intensities, because tinnitus cannot be increased or diminished in intensity at will like externally applied sounds; but rough monaural measurements may be made, if the patient estimates the loudness necessary to double the intensity of the tinnitus, or the intensity necessary to just mask the tinnitus (Fletcher, Steinberg and Wegel); however, one may apply like frequencies alternately to two ears, one of which harbors a tinnitus, and balance the loudness of these tones at higher and higher levels of intensity. If this is done, certain differential diagnostic phenomena may be observed.

The presence of tinnitus affects the binaural loudness balance up to a certain point, at the different frequencies, in much the same way as an applied band of like frequencies and intensities; in other words, it has an effect similar to lesions affecting comparable elements of the neural apparatus of hearing (see Fig. 3a). The two ears will balance with equal loudness at and above a critical bilaterally equal intensity. In pure obstructive deafness with tinnitus, there is a rapid recruitment of loudness in the ear harboring the tinnitus up to the point of loudness where the testing sound eliminates the deafening effect of the tinnitus. The recruitment ceases at the intensity (in the tinnitus ear) which is the measure of the obstructive loss (see Fig. 3c). The tinnitus thereafter cannot operate as a recruitment factor; and (in the absence

of nerve deafness) there is no further change in the relative number of decibels required for loudness balance in the two ears as intensities are increased. Thereafter, the balance is stabilized as in ears with a monaural or unequal binaural uncomplicated impedance (obstructive) deafness. Thereafter, the two ears do not vary in the number of decibels separating their loudness levels.

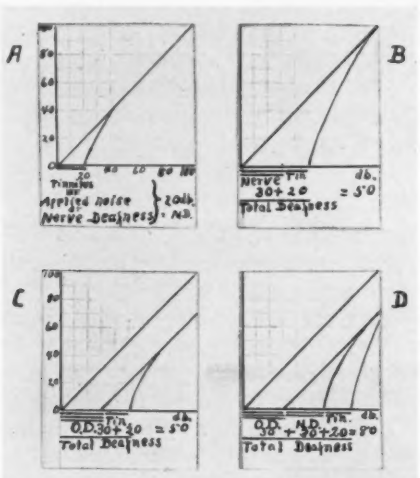

Fig. 3. The effect on the alternate binaural loudness balance of deafness from middle-ear (impedance), from inner-ear (neural) lesions, and from tinnitus or simulated tinnitus from externally applied sounds: (a) Effect of tinnitus, or simulated tinnitus from applied sounds (or the neural lesions causing the tinnitus. (b) Ditto, plus neural lesions causing deafness without tinnitus. (c) Ditto, plus middle-ear impedance lesions. (d) Ditto, plus (b) plus (c). (o.d.) = Obstructive deafness. (n.d.) = Nerve deafness.

On the other hand, in pure nerve deafness, even after the recruitment dependent upon the tinnitus as such is completed, it continues toward a more equal bilateral loudness level. The two ears tend to coincide more and more in the estimation of equal intensities as intensities are raised (see Fig. 3b). If from the total neural deafness is deducted the number of decibels indicating the loudness of the tinnitus, the remainder will be the amount of the nerve deafness as such; *i.e.*, the deafness caused by the neural lesions without the presence of tinnitus.

Whether tinnitus is present only coincidentally or as a result of impedance (obstructive) or nerve lesions, or both of these,

the above tests may serve to differentiate the amount of deafness referable to each of these (see Fig. 3d).

Someone will say, "What difference does it make? Tinnitus alone causes deafness, whether it originates from within or without the ear, or whether or not it is due to mechanical or neural lesions." Up to a certain point this is true; but the tests I have proposed make it possible not only to measure the loudness and effect of a tinnitus as such, but to determine if and to what extent it is accompanied by an obstructive or a neural lesion. In other words, to measure the loudness of the tinnitus and determine how much, as such, it contributes to the deafness. Again, I state that, with certain limitations, this is possible whether or not the tinnitus is of impedance (obstructive), or of neural lesion origin, or is simulated by an applied frequency.

HEARING AIDS.

It is apparent that many of these normal and pathologic peculiarities of auditory, acoustic and neural reactions are pertinent to the prescribing of hearing aids. One great fault of the hearing aid is that, although it may bring up the volume of sound to useful intensities for nearby sounds, it lacks the ability possessed by the normal ear to maintain automatically the normal proportion of sound with increasing distance. In other words, *no aid restores the normal range of hearing.*

The first requisite is to bring to the ear a sound level which is both sufficient for good hearing and agreeable to the hearer; not too loud and not too faint; the vowels and consonant sounds in proper proportions. Quality is important, but the ear is so accustomed to hearing voices of various pitches and timbre, and poor quality in telephone and loud speaker, to rasping, falsetto, booming or squealing voices, that it automatically tends to interpret all such in terms of ordinary speech, and does not permit them to interfere much with understanding. There is, fortunately then, some leeway for all concerned.

In pure obstructive deafness, the main objective is comparatively simple to attain. The ear should receive from 15 to 50 dcb. above speech minimum audibility, depending upon distance and the amount of surrounding noise. To do this in nerve deafness is unsatisfactory, because here the loudness levels are influenced by the ratio of the recruitment phenome-

non, and this in turn varies with the position and extent of the lesions causing the deafness.

If recruitment of loudness with intensity is slow, *i.e.*, if the deafened ear does not recruit at least 10 dcb. until 30 dcb. are added to its minimum audible intensity, it will require more amplification in the frequencies thus affected than when the recruitment is rapid; *i.e.*, where a near normal loudness

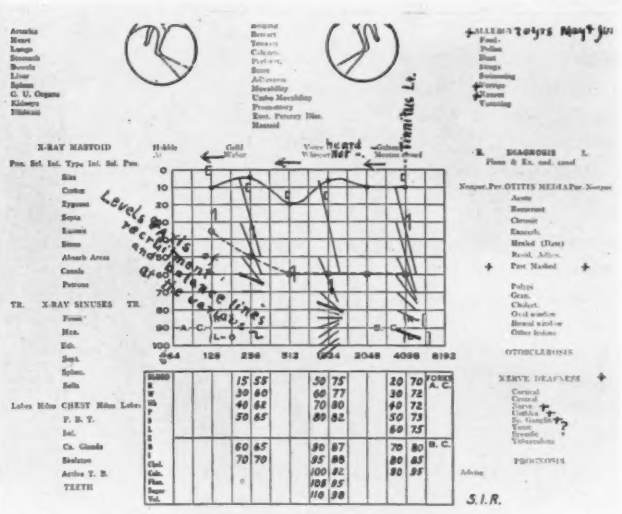

Fig. 4. The short straight lines crossing the ordinates at each frequency indicate the equal balance levels between the right and left ears at different degrees of loudness above threshold. The lines crossing the 1,024 frequency ordinate show an equal loudness balance at 83 dcb. above normal threshold; but louder sounds reveal a reversal in the slope, which means that the worse (left) ear is sensing these very loud sounds as even louder than in the normal (right) ear. This I call the phenomenon of over-recruitment of loudness.

is attained at 10 or 15 dcb. above minimum audibility. When slow, 30 dcb. or more amplification must be added for the ear to pick up 10 dcb. over the added loudness. When rapid, 10 or 15 dcb. added to the threshold intensity is all that is necessary for normal loudness.

If obstructive deafness exists coincidentally with nerve deafness, it is necessary to add sufficient amplification to make up the loss caused by the impedance (obstructive) lesions, and then to control over-loudness according to the

ratio of recruitment. In other words, in obstructive deafness it is possible by selective amplification to replace the lost intensity in the speech range, whereas in nerve deafness no such simple correction is possible.

It is at present practically impossible with a light portable hearing aid to bring up the high frequencies to near normal loudness without also bringing up the lower frequencies, to over-accentuate the hearing for low tones, and produce a sound something like some of the old, badly designed radios. Bass drums and bass viols and vowel sounds boom out and tend to drown out the middle and higher tones (and consonants). Theoretically, it is possible to remedy such defects in hearing aids, but in many instances selective amplification is not sufficient without selective repression.

One of the most disagreeable defects in all electrical hearing aids is the overpowering noise from nearby undesirable sounds; the roar from many voices, which masks out the ordinary speech of anyone not very close to the listener; the blasts from nearby sound when the aid is adjusted for distant speech sounds.

Environmental noise may be in a way advantageous in obstructive deafness, because the deafness cuts down the masking noise; the person speaking automatically approaches the listener, or is speaking so much louder than usual in order to make himself heard that his voice reaches the deafened listener with great intensity; but the hearing aid does not automatically cut down the disagreeable more than the agreeable sounds.

Try listening to conversation under the elevated railroad while a train is passing and you will demonstrate these phenomena, which, of course, have been known but not fully understood for ages. While using a portable aid in the theatre, if someone nearby claps his hands it may sound as if the building is falling down. These blasts of sound may be not only disagreeable but dangerous with uncomplicated nerve deafness, because the vibrations reach the inner ear with as much intensity as in a normal ear. Drum perforations and wide open Eustachian tubes also lessen for the lower tones the protective air buffer effect of a normal closed middle ear.

Some sort of shock absorber is indicated, some device which will allow free activation of the receiver up to a certain inten-

sity and no more. This may be supplied mechanically* and electrically. A "Varistor," or some modification or new device to accomplish this end should be incorporated in the hearing aid.

A NEW PHENOMENON.

Making it a practice to search for the presence of the recruitment effect has revealed in some ears with nerve deafness that very loud sounds appear even louder to a deaf than to a better hearing ear. Lorente de Nó's researches made it possible to explain the recruitment of loudness phenomena on the basis of three facts: *1.* Stimulation of a neuron requires that several impulses impinge upon it within a very short time (less than 500 microseconds); *2.* the frequency of the impulses conducted by a nerve fibre cannot be greater than a certain limit characteristic for each fibre (approximately 800 per second); *3.* the strength of stimulation of the nerve centres is within certain limits proportional to the number of afferent impulses they receive per unit time. Consequently, if one neuron or group of neurons are missing or ineffective for any reason, sufficient loudness will bring others into activation and finally reach a point where as many nerve impulses are sent to the higher sensory stations in the brain from the diseased (or occupied) as from the normal (or unoccupied) side. This accounts for a full recruitment of loudness, but not for an over-recruitment.

The over-recruitment phenomenon is explained by Dr. Lorente de Nó as follows:

"Over-recruitment indicates that beyond a certain tone intensity a larger number of impulses are sent to the cerebral cortex from the diseased than from the normal side. This may be related to the fact that the transmission of impulses through the primary acoustic nuclei is not continuous; it is interrupted by phases of depressed excitability of the nuclei.† This phenomenon is similar to the 'inhibition' of the spinal flexor reflex and indicates that the transmission of impulses through the acoustic nuclei is similar to transmission through other central pathways, excitation of determined channels being accompanied by inhibition of other channels. Only by means of inhibition can the impulses be prevented from enter-

*E. P. Fowler: Thesis for U. S. Army, 1934.
†Cf. The Laryngoscope, 45:573-595, 1935.

ing into parallel channels to produce diffuse stimulation. Obviously, if the impulses were not kept within selected channels, 'localization' of peripheral stimuli would be impossible and the sensation of pitch could not be explained by 'localization' of tones on the basilar membranes.

"Central inhibition requires most accurate timing of arrival of impulses* and, therefore, it is thinkable that when the acoustic nerve or the organ of Corti is diseased, the inhibitory mechanisms in the acoustic centres are less effective than under normal conditions, and when overloaded allow the passage of impulses through abnormal channels, so that the cerebral cortex receives a larger number of impulses than it would receive under normal conditions. The loudness of the perceived tone must be greater, but at the same time, if this explanation is true, the character of the tone must be changed and the patient cannot hear tones of very high intensity as pure tones, but rather as mixed tones, or even as noises."

This is exactly what has been observed clinically.

140 East 54th Street.

DISCUSSION.

Dr. Max A. Goldstein (St. Louis): For routine examination and functional tests of hearing with the audiometer, the threshold of hearing as now arranged in most of these apparatus at our disposal serves the purpose of differentiating the various types of deafness; but in more profound hearing depreciation as, for instance, in the deaf child, the construction of the audiometer should include decibels of much higher intensity in tone frequencies of the speech range to a much higher degree than is now furnished in most audiometers at our disposal.

As to the tone frequency problem in the hearing range, I would call attention to the fact that most of our audiometers are constructed with a low limit at 128 d.v. The normal ear can readily distinguish a tone frequency at 32 d.v., and if we are to use the complete audiometric scale as a diagnostic aid in determining the hearing of low frequencies, the audiometer should include at least 64 d.v. of the tone scale.

It is possible to have a patient with an incipient bone conduction defect who will hear 128 d.v. distinctly, and if there is no lower limit for testing it will be impossible to determine whether he hears 64 d.v. or not.

*Cf. Analysis of the Activity of Chains of Internuncial Neurons. Trans. Neurophysiol., 1938 (in the press).

I have urged the committee of physicists and otologists working in conjunction with the American Bureau of Standards in the perfection of the audiometer for its application in clinical otology to include 64 d.v. as the low limit of tone frequency in all audiometers.

After all, the audiometer is not used exclusively as an instrument for determining what kind of hearing aid we should prescribe for an individual patient; it is intended much more specifically for use by clinicians in otology to determine the type of deafness and for differential diagnosis of types, and we should have the privilege of demanding what we have always held a necessary adjunct to our diagnosis in functional tests as educed from our clinical experience in the past — that for universal use, an audiometer should include tone frequencies from 64 d.v. to 8,192 d.v. and beyond, as we have always had the privileg of measurement by numerous other instruments, including tuning forks, Galton whistle and monochords.

The audiometer is designed not only for the acoustic laboratory but also for the use of the clinical otologist. (Applause.)

Dr. Harvey Fletcher (New York): I found Dr. Fowler's paper a very interesting one and I feel that he is to be complimented on the new concepts he has presented.

I was especially interested in his suggestions for measuring tinnitus and in his method for determining the true threshold for bone conduction. You will recall that he showed us a slide of an audiogram, in which the bone conduction was normal at 128 and 256 cycles per second and then dropped very markedly at frequencies of 512 and above. Dr. Fowler pointed out how the readings of "normal" at 128 and 256 were subsequently shown to be spurious. He determined this by a clever test. He made the patient *hear*, not merely signal, because Dr. Fowler had the patient balance the tone by bone and air conduction at different levels above threshold. Then, for example, when the air conduction tone was only 5 dcb. above the threshold value, the bone conduction reading was found to be 40 dcb. above the spurious threshold.

Obviously, the patient was not hearing in the original test at 128 and 256 cycles per second, when he signaled correctly at a zero hearing loss setting. He may have felt the vibration. I can readily understand such tactile perceptibility if the patient were a so-called "deaf" child who had received considerable training in a school for the deaf, where the tactile sense is used to supplement the residual hearing.

We have long questioned the validity of bone conduction tests at low frequencies because of the possibility that the vibration might be felt, rather than heard. Dr. Fowler has given us a method of determining the true bone conduction threshold.

XANTHOMATOSIS (SCHULLER-CHRISTIAN'S DISEASE). A REPORT OF A CASE WITH RADIOSENSITIVE PATHOLOGY IN THE MASTOID.*

DR. JOHN J. SHEA, Memphis.

Roentgen therapy has been advised for the treatment of pathology affecting the middle ear and mastoid, ranging from simple catarrhal conditions to acute suppurative involvements.[1, 2] The enthusiastic reporters have credited it with miraculous powers, but the clear thinking otologist has failed to endorse this enthusiasm; however, in Schuller-Christian's disease the action of deep X-ray therapy is marvelous.

The literature of the past 10 years records many worthy papers upon xanthomatosis,[6 to 16] with a clinical syndrome of a triad of bony defects of the skull, diabetes insipidus and exophthalmos. Hand,[3] in 1893, reported a lad, age 3 years, who presented exophthalmos, polyuria and a cystic degeneration of the right parietal bone. Schuller[4] later reported two cases with the triad, and Christian,[5] in 1919, attracted the attention of American physicians to the work of Schuller and reported an interesting case. Rowland reviewed the literature and applied the term xanthomatosis to the condition.

Xanthomatosis differs from Gaucher's disease, in that the latter involves the spleen, is familial and most frequently occurs in female children. Niemann-Pick's disease is a rapidly fatal xanthomatosis, with an enlargement of both liver and the spleen, affecting most frequently children of the Hebrew race.

PATHOLOGY.

The pathology is that of a disturbance of the lipoid metabolism, especially of cholesterol, limiting its secretion and increasing the storage of lipoid material in the region affected. Rowland classed it as a systemic condition with dermatological

*Read before the Seventy-first Annual Meeting of the American Otological Society, Inc., Atlantic City, May 6, 1938.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1938.

manifestations, and its lesions are the result of the phagocytic action of the reticuloendothelial system. The same author claims that lipoids, when in excess in the body fluids, become pathogenic to the individual; they irritate the vessel walls and are followed by perivascular-cell infiltration with a degeneration of the part affected.

REPORT OF A CASE.

M. F., white male, age 16 years, was seen in consultation with Dr. H. W. Greenburgh. His chief complaints were an

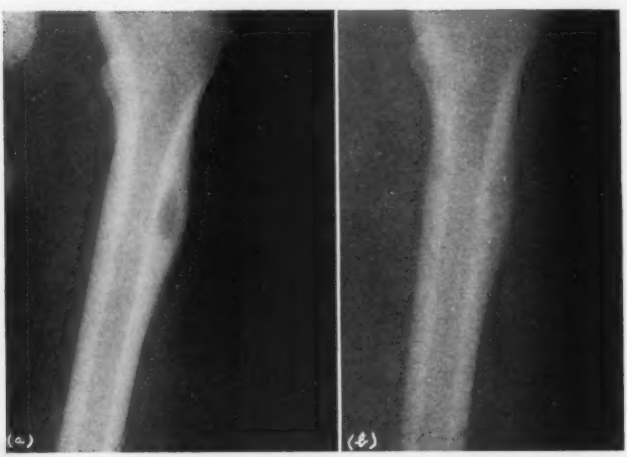

Fig. 1. (a) Fusiform cyst of the femur. (b) Cyst has filled-in after radiation.

impairment of hearing in the right ear and an acute vertigo upon arising and relieved by standing upright. The past history of the lad included measles and mumps. His growth had been normal and his habits regular. The family history did not include any instance of diabetes, tuberculosis, malignancy or endocrine diseases.

During October, 1936, the lad complained of pain in the left hip. Examination by Dr. Willis C. Campbell was reported as follows: "The patient complained of pain and tenderness of left upper thigh. History given: One month previously, he struck his thigh against the corner of a table. This was followed by tenderness, which subsided in a few days, but from

which he has never completely recovered. There have never been any acute symptoms, such as fever, local heat or redness. Skeletal examination: The patient shows an old rachitic deformity of the chest. There is no external abnormality noted in the thigh, except a point of slight tenderness to deep pressure over the anterolateral aspect of the thigh about four inches below the greater trochanter.

"Roentgenological study revealed a fusiform cyst in the femur cortex below the greater trochanter, but not encroaching upon the medulla and with slight elevation of the periosteum. The lad was studied with the fluoroscope, but no other cyst was found.

"Laboratory examination shows a negative Wassermann and Kahn, with the Meinicke 2+.

"Several diagnoses were considered at this time: *1.* A low-grade infectious process simulating osteomyelitis. *2.* Specific luetic osteitis. *3.* Malignant tumor.

"Subsequent X-rays, taken Oct. 28, 1936, showed no material change from the previous reading except for slight progression in the size of the lesion. An exploratory operation was advised at this time, because of failure to respond to antiluetic treatment and the possibility of an early malignancy. Operation was refused, so deep Roentgen therapy (Dr. W. S. Lawrence) and a tonic were administered. The lesion subsided and the symptoms disappeared."

EXAMINATION.

Examination of his nose and throat was essentially negative, except that the lad had a mild cold, with the usual congested nasal membranes and inflamed lymphoid deposits of the pharynx. The right ear appeared normal and, through a very thin drum, the usual contents of the middle ear were visible. The hearing test was as follows:

	Low Forks	High	Rinné	Bone Cond.	Whisper
Right	32	C^4	+	Normal	20 ft.
Left	32	C^4	+	Normal	20 ft.

The Barany tests were reduced to the caloric technique. The lad could not tolerate rotation. The left ear reacted in 50 seconds and the right ear responded violently before 30 seconds of irrigation. Roentgenological examination revealed a very

peculiar arrangement of the right temporal bone. The left temporal bone was extensively pneumatized and appeared of normal density. The right temporal bone presented an extensive loss of intracellular walls with a cystic appearance of the zygomatic region extending onto the squamous portion. In the Stenver's position, there was a loss of the continuity of the upper border of the petrous portion with a rarefied area above it. This loss of bone structure in the petrous process made the capsule of the labyrinth unusually visible.

Diagnosis: The temporal bone lesion was suspected of being a metastasis of the cystic tumor in the thigh. Another complete physical was done, which was reported normal, except for a mild hypochromic anemia and a cholesterol of =259 mgm. per cent.

Treatment: Roentgen therapy was again tried upon the advice of Dr. Lawrence, who first suggested that the condition was possibly of a Schuller-Christian type of xanthomatosis. Under treatment, the dizziness disappeared and the normal markings returned in the Roentgenogram of the right temporal bone.

Subsequent History: During the late spring, the lad complained of polydypsia, polyuria, general weakness and a loss of weight, with pains in the chest at night. Blood pressure was 140/70 and a general physical was negative, except for the presence of rales at the base of both lungs. Roentgenological study of the chest revealed a diffuse cystic degeneration of both lungs and similar changes in the mandibles. The urinary output was 6,000 cc. daily, with 1,004 specific gravity, and the tests for sugar were negative.

The lad entered Johns Hopkins Hospital under the care of Dr. W. T. Longcope, whose additional findings were: "He had no trouble until four weeks ago and gained weight. When on a trip to New York he noticed stiffness in the left hip and developed a cough with pain in the chest. In two days' time both the leg and the chest got better and since that time he has been no worse and no better. Just before this episode four weeks ago, patient had had marked swelling in a lower left premolar area, thought to be an abscess, but because of Vincent's infection, the tooth was not extracted. Patient has lost five pounds in the last three weeks; weighed 118 last fall, but

since that time has grown quite a bit. Patient has developed polyuria, 10 to 12 pints over one 24-hour period, and has polydypsia to some extent, all this coming on after X-ray treatment to the head.

"Physical examination revealed a tall, thin lad, rather a long face, jaws being rather markedly elongated. There was no exophthalmos. No features to suggest acromegaly. Fundi

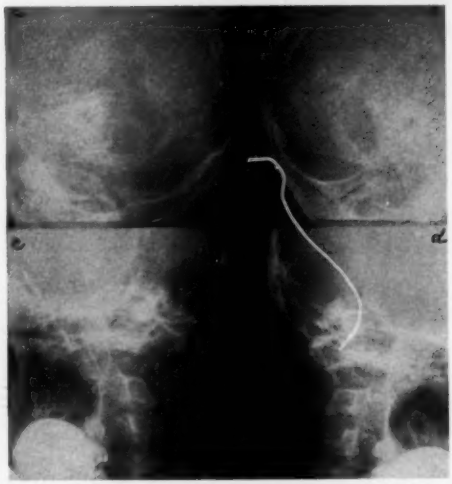

Fig. 2. (a) Right mastoid. (b) Left mastoid. (c) Right mastoid, Stenver's position. (d) Left mastoid, Stenver's position.

showed no abnormalities. Apparently there is suppurative gingivitis, partial upper denture. Area in lower jaw, where swelling was before entry, is quite negative in appearance. Thyroid not remarkable, small gland under right maxilla just felt. Thorax expansion equal. Lungs were clear to percussion and auscultation. The heart was not enlarged. The examination was fairly negative except for a split second sound and rather marked sinus arrhythmia. Blood pressure, 110/72. Abdomen was soft, there were no masses, liver not felt. The site of the old cyst in the right upper field was fairly negative, possibly a little irregularity in the left upper femur. There was no pigmentation anywhere on the body.

"R.B.C., 5,670,000; hgb., 13 g.; vol. packed R.B.C., 42.5; W.B.C., 5,700; sedimentation rate corrected, 27; ict. index, —2; marked microcytosis, marked poikilocytosis. Differential count: Juv. neutro., 2; seg. neutro., 59; eosino., 4; lymphocytes, 26; monocytes, 6.

"N.P.N., 24 mgm. per cent; sugar, 86 mgm. per cent; ca.=9.3 mg. per cent; P.=5.0 mgm. per cent; chol.=188 mgm. per cent; R.I.=6.6 mgm. per cent protein.

"The Wassermann was negative.

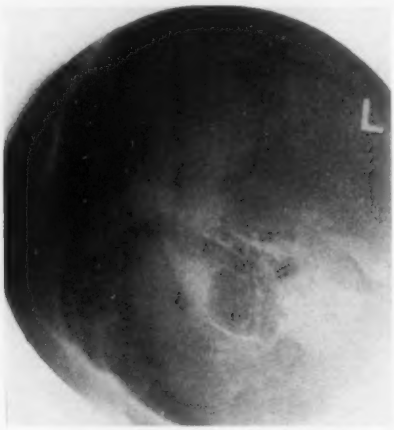

Fig. 3. A large cell without pathology.

"Repeated blood examination: W.B.C., 5,300; differential count: seg. neutro., 74 per cent; juv. neutro., 5 per cent; lymphocytes, 16 per cent; eosino., 3 per cent; monocytes, 2 per cent.

"A sugar tolerance test was done. Fasting specimen, 86 mgm. per cent; one-half hour after glucose, hct., 109; subsequent figures: 97, 74, 68 and 69.

"All urine examinations were negative. It was revealed that the volumes on three single specimens taken an hour apart were, respectively, 450 cc., 350 cc. and 325 cc.

"The original X-rays were obtained and revealed a cortical cyst in the left femur and a similar cystic area in the skull.

Subsequent plates revealed complete healing of the lesion. The lesions of lungs found in X-ray plates were thought interesting. Patient was placed on nasal insufflation of powdered pituitrin, posterior lobe, and this resulted in a marked effect on the urinary output. The day before the pituitrin had been given, the patient had 9,200 cc. of fluid and had put out 11,850. The day after the pituitrin treatment, the output was 5,700; the intake, 9,950. He was tried one day on pituitary jelly, but since he decided to go home and the urinary output on this day was 7,375 cc., as compared with 4,625 cc. on the

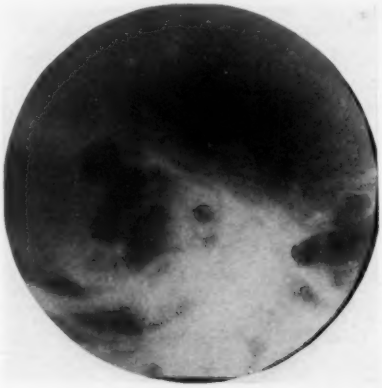

Fig. 4. Cholesteatoma.

day of pituitrin, it was decided that the patient should be discharged by using the nasal pituitrin by powdered inhalation and to return later for a check-up. Repeated W.B.C., July 2, 1937, was 9,700. In view of the findings on X-ray, *viz.*, extensive linear infiltration of the lungs, changes sometimes seen in Xanthomatosis; large areas of absorption of the mandible in the region of the lower molars, it was felt that the diagnosis was probably Christian's disease; diabetes insipidus; manifestations being bone cysts."

DIFFERENTIAL DIAGNOSIS.

Roentgenological: The picture of a cystic degeneration of a temporal bone could be a developmental defect, as illustrated in Fig. 3, or an extensive destruction by a cholesteatoma, as

illustrated in Fig. 4, but these two conditions are limited to the mastoid process and do not involve the petrous portion.

Chemical: The stored lipoid has a high cholesterol or cholesterol ester content (Epstein and Lorenz[16]).

Surgical Experience: Chester and Kugel[17] reported an incident of surgery upon a mastoid, the seat of a xanthomatosis, which continued to discharge for more than two years until death, even with the aid of Roentgen therapy. Dr. Allen B. Potter, in a personal communication, kindly allowed me to review the history of a patient, which he will report in the near future.

"Child was a male, age 1 year and 9 months, with a history of discharging ears for one year. Physical examination revealed a yellowish discharge, with a very offensive odor filling both external auditory canals. At operation, the right mastoid contained a mass of brownish-red granulation tissue studded with small yellow granules. One month after discharge from the hospital, the child developed two nodules of the skull. The child lived for nine months and at death the mastoid wound was covered by a necrotic membrane, and the two nodules of the skull were found to be 'collections of yellow material with a modern amount of connective tissue replacing the outer table of the cranial bone.' The mastoid and petrous portion of the left temporal bone apparently have been completely replaced by this yellowish tumor and the right mastoid and petrous portion infiltrated with it."

CONCLUSIONS.

1. The pathology of Schuller-Christian's disease is radiosensitive.

2. Further study is necessary to discover a means of readjusting the disturbed lipoid metabolism.

3. Surgical interference should be avoided.

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DISCUSSION.

DR. LEROY A. SCHALL (Boston): During the past 10 years, as the author states, there have been worthy papers upon xanthomatosis. These cases have been studied and reported by a general practitioner, Roentgenologists, internists, a pediatrician, physiologic chemists, ophthalmologists, a dermatolo-

gist, a neurologist, a neurosurgeon and pathologists. It is well that the disease is now being recognized by the otologists. Involvement of the mastoid has been reported by Sosman, citing a case of Vogt's, by Chester and Kugel, by Wood and now by Shea.

Five clinical entities are recognized as coming under the heading of xanthomatosis; namely, Gaucher's disease, Niemann-Pick's disease, Schuller-Christian's disease, the xanthomas occurring in icterus, diabetes, and pregnancy, and the so-called essential xanthomatosis. Of these, the Schuller-Christian's disease is of otologic interest, as it is characterized by granulomatous deposits in the bones of the skull, occasionally in other bones, with frequent association with diabetes insipidus, exophthalmos and gingivitis.

As the signs and symptoms of this disease depend upon the location of the deposit, we may expect to occasionally find mastoid involvement. Of the 12 cases studied at the Children's Hospital, three showed this involvement, simulating acute mastoiditis. One patient had a mastoidectomy previous to admittance, while the other two presented aural discharge, mastoid tenderness and the X-ray finding of mastoid destruction.

In judging the efficiency of any type of treatment, it must be remembered that cases of spontaneous remission have been reported. As most of the evidence points to an abnormal level of lipoids, particularly cholesterol, an attempt to control the increase of lipoids, by a low fat diet, is indicated. As many of the cases present diabetes insipidus, solution of pituitary, either subcutaneously or by nasal spray, has been used to advantage. The local deposits in the bones have shown uniformly beneficial results when treated by the Roentgen ray. It must be assumed that the irradiation kills the already distended and fat-laden histiocytes, liberating the lipoids, permitting the individual lesion to heal. The end-result after Roentgen ray therapy, as was shown in a case studied at the Massachusetts General Hospital, is a non-specific fibrosis.

I wish to emphasize Dr. Shea's conclusion by repeating "that mastoid surgery has no place in the treatment of this disease."

REPORT ON THE RESULT OF SUBMITTING HUMAN SUBJECTS TO ROTATION.*

DR. EDUARDO R. ARELLANO, Havana.

The title of my article appears in the program as being related to human subjects, but as a matter of fact we have experimented only on dogs.

We have delabyrinthized dogs on one side — always the left; then, placing, in turn, each one of the right canals in the horizontal plane; the right horizontal, then the right anterior vertical, lastly the right posterior vertical — we rotated the dog and took moving pictures of the nystagmus.

We have made a good many experiments, and now we know much better than before how any single mammalian canal is affected by rotation. We will not go into detail but will show the method and mention just one or two of the chief results.

In each experiment the dog was bandaged like a mummy and placed on the revolving platform. A moving picture camera in front of the dog's head was used to record the nystagmus during rotation, with electric lamps behind the camera for the illumination. In turning we always followed a certain order: First, the platform was placed horizontally in order to stimulate the right horizontal canal, then, vertically, with an inclination of 45° towards one side for the right anterior vertical; then inverted to the other side for the other vertical; in each case counter-clockwise, then clockwise. So each experiment has six stages. These rotations were carried out at various speeds and for a period of 20″ each time. Always the same speed in any one set of experiments.

The following illustrations show the procedure:

a. The turntable, to show the dog box, camera and electric lamps.

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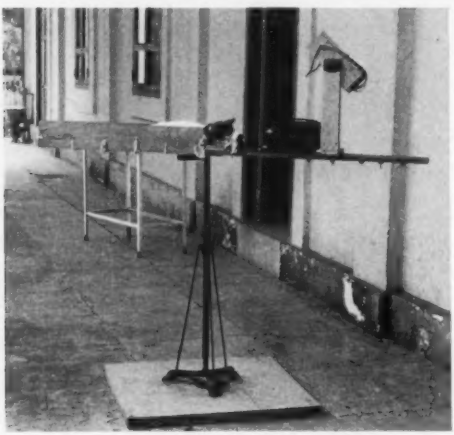
b. The immobilization, dog tied down like a mummy.

c. Dog on turntable, showing position of camera in front of the dog's snout.



d. Position for vertical canals, for right anterior vertical canal. Invert for right posterior vertical.

Our findings were:

1. Mammalian canals, unlike those of lower vertebrates, may be stimulated by a movement in either direction. A film is shown to demonstrate this assertion.

2. Each canal of the three is more easily excited by rotation in one direction than in the opposite direction. A horizontal canal responds best to an ampulla-petal movement. An anterior vertical to an ampulla-fugal movement. And a posterior vertical to an ampulla-petal movement. A film is shown to demonstrate this finding.

As may be seen, the horizontal and anterior vertical canals respond according to Ewald's rule, but the posterior vertical respond better to an ampulla-petal movement, or contrary to Ewald's findings. This has been tested in many experiments in both phases of rotation, for which we must accept it, at least in dogs.

3. The nystagmus produced during rotation is due to excitation arising in the labyrinth. One might think that some of the results seen in these films are due to the dog trying to fix its gaze and then quickly shifting. In actual truth, voluntary fixation, if it does anything, interferes with nystagmus. Take both labyrinths away and see what the result is: A film of a bilaterally delabyrinthized dog is shown in which no nystagmus appears during rotation. After seeing this film, I think you will agree with us that nystagmus in a dog is not visual; it needs the labyrinths and the canals.

Time does not allow me to mention other interesting results but they will be referred when published.

Malecon 250.

DISCUSSION.

PROF. JOHN TAIT (Montreal): There is one little correction that I want to make: Dr. Mosher was under the impression that this work was done under my superintendence. I wish it had been, because, in my estimation, it is very important work; but it was done independently by Dr. Arellano and Dr. Abalo working in Cuba, apart from any external resources and apart largely from the literature dealing with the subject. The work is entirely their own.

They began some years ago, and let me here exhibit some of the films that these men have taken in their investigations (shows numerous cartons containing rolls of film). I think it is only fair that we should get some conception of the extent of their work, the expense to which they have gone in doing it, and of the time that has been consumed in its execution. When I show this great collection, let me say that these are not all the films taken. I thought they were when I saw them the other day, but Dr. Arellano has a great many more with him. These transported films are the good ones, that can be exhibited and shown. He brought them in order that anyone interested in any particular detail might have a chance to view the special point at issue. He left less successful films at home.

While he has elected to present only his method, leaving himself little or no time to indicate the results they obtained, the purpose of the investigation was, of course, the collection of numerical data. Though many different dogs were used, these data turned out to be, in their relationship to each other, extraordinarily regular and consistent. As you can understand, too, from a collection of films such as they have assembled they have been able to make an unusually large number of counts. In the fewest possible words I am going to attempt to give you some of the chief conclusions that they derive from analysis of their data, and I shall use the blackboard for that purpose.

First of all, we will take the case of the dogs that were unilaterally delabyrinthized. A dog of this kind has only three solitary canals, namely, on the right side. When each, in turn, is laid in the proper (horizontal) plane, one can be reasonably sure that, in the rotation, only that particular canal that is horizontally placed for the time being is being stimulated.

At any selected speed of rotation any single canal of a unilaterally delabyrinthized dog is responsible for exciting a certain number of nystagmic

beats. These numbers differ according as they are counted in the rotational or in the postrotational phase. They similarly differ according to the original rotation occurring in an ampulla-leading ("fugal") or an ampulla-trailing ("petal") sense. By averaging the results taken at the three most commonly used speeds, one obtains what the authors, for convenience, name "the nystagmic number" for each canal of the three, according to whether the corresponding endolymph movement is fugal or petal, respectively. These numbers are as follows:

THE NYSTAGMIC NUMBERS.

	Ampulla-leading (Fugal)	Ampulla-trailing (Petal)
Horizontal	17	24
Anterior vertical	16	3
Posterior vertical	5½	8

From the table one can see at once that each canal, as Dr. Arellano has said, has its preferential sense. Thus, the preferential sense of a horizontal is ampulla-trailing, that of an anterior vertical is ampulla-leading. This is similar to what Ewald found in the case of a bird, but note the preferential sense of a posterior canal of the dog. It is ampulla-trailing! This result, so far as the mammal goes, is entirely new. What is more, it is a perfectly dependable conclusion in the case of the dog. This means that in any nose-down rotation of the dog's head, both the posterior and the anterior vertical canals are more active in exciting nystagmus than in the nose-up rotation. That is the first important point.

The second point, with which I am practically going to finish, is that they not only determined the "nystagmic number" for each canal taken separately, but they went ahead and, for sake of comparison, got corresponding figures for normal dogs. Of course, from normal dogs one gets a greater number of nystagmic beats each time, because then, you see, two synergic canals are working.

I am not going to write these new numbers down, but what they did was to express the original nystagmic numbers as a percentage ratio of the new numbers obtained from normal dogs. In this way they obtained what they call the "percentage activity ratio" of each canal, or, if you like, the proportion of nystagmic beats contributed by one canal to the total number due to stimulation of a synergic pair. Let us look at the figures.

PERCENTAGE ACTIVITY RATIO.

	Ampulla-leading	Ampulla-trailing
Horizontal	43%	60%
Anterior vertical	57%	11%
Posterior vertical	19%	28%

Now, here is something rather interesting. Add up 43 and 60, the figures for a horizontal canal, and you get 103. That is practically 100 per cent, which is just what it ought to be. Now, add up 57 and 28 (remember that in this case we have to add the anterior with the posterior on the other side). The sum is 85, not 100 per cent. There is a shortage of 15 per cent. Add up 11 and 19 and you get only 30 per cent, still greater shortage.

How do they account for this curious feature in the case of the vertical as compared with the horizontal canals? Here is what they do. Dr. McNally and I, working on canals, eventually concluded that, whereas the utricular maculae have nothing to do with the horizontal canals, they stand to come into play in almost all rotational movements that excite the vertical canals.

To put the matter briefly, the authors think it possible, or even likely, that in what would correspond to tipping movements of the head, the utricular maculae, as well as the canals, make their own separate contribution to the total number of nystagmic beats.

Last of all — and this is the point that will appeal to a clinical audience, though Dr. Arellano in his own account elected to omit it — the Cuban workers have made similar experiments on two human subjects with a unilateral loss of the labyrinth. From these two persons they have obtained provisional "nystagmic numbers" for the rotations.

Suppose that one had the nystagmic numbers in a sufficient number of cases, something comparable to what I have shown here in this suitcase. It then would be a simple matter, counting the number of postrotational beats, to diagnose a lesion of any one single canal itself. The caloric test can already tell us of the destruction of all the canals of any one side. But what is soon going to be possible, notwithstanding the fact that any one mammalian canal responds in two senses, is sharp and clear diagnosis of destruction of any one of the six, taken separately. (Applause.)

THE ENDOLYMPH.*†

DR. L. M. POLVOGT, Baltimore.

The structure of the membranous labyrinth is described in all textbooks of anatomy and otology, but very little is said about the origin, direction of flow, pressure or chemical composition of its fluids. We know that the perilymphatic space communicates freely with the subarachnoid space of the posterior fossa and that perilymph has the same chemical composition as cerebrospinal fluid. The endolymph, however, is in a closed system, which extends from the apical end of the cochlear duct to the saccus endolymphaticus, which lies in the dura near the lateral sinus. The entire system is so small that it contains only a few drops of endolymph. An analysis of this minute quantity could be made with microchemical methods if it were possible to obtain a specimen free from contamination with blood, cerebrospinal fluid or perilymph.

There is every reason to believe that the endolymph is of prime importance in the normal functioning of the auditory and vestibular apparatus. The end-organs in the vestibular system have a blood supply, but no one has demonstrated blood vessels in the end-organs of the cochlear nerve. It is thought that oxygen and the necessary mineral salts reach the hair cells of Corti's organ by way of the endolymph, and that the by-products of their metabolic activity are carried off by this fluid.

The ingenious experiments of Guild on the origin and direction of flow of endolymph suggest that in the cochlea this fluid is secreted by the stria vascularis, just as the choroid plexus in the brain secretes cerebrospinal fluid. Anatomical and experimental studies would indicate that a constant secretion of endolymph is essential for normal hearing, but, to our surprise, the correlation of histologic appearance and function in ears whose hearing was tested a short time before

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†From the Otological Research Laboratory of the Johns Hopkins University, Baltimore.

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death shows that extensive areas of strial atrophy do not cause impaired hearing. The problem cannot be solved by such simple methods, and our failure emphasizes the need for the correlation of hearing acuity with chemical studies of the endolymph. Chemical analysis alone without exact information about the hearing would be of little value.

The canalis reuniens connects the cochlear and vestibular endolymphatic spaces, and Guild's experiments show that substances injected into the cochlear duct are found a few hours later in the saccus endolymphaticus. Histologic study of temporal bones of man also show pigment cells from the stria vascularis in the saccus endolymphaticus. All information we have about endolymph indicates that it is secreted by the stria vascularis in the cochlea and by somewhat similar structures near the vestibular end-organs in the sacculus, utriculus and semicircular canals; that its direction of flow is from the cochlear and vestibular apparatus through the ductus endolymphaticus to the saccus endolymphaticus, and that it finally passes through the walls of the saccus into the venous system. We know nothing about clinical conditions which may obstruct the flow of endolymph, or the symptoms resulting from interference with its circulation.

The object of this paper is to emphasize the importance of the endolymph to hearing and to equilibrium, and the need for investigation of the labyrinthine fluid in normal and diseased ears. Crowe has recently shown that there are many reasons to suspect that a physical or chemical disorder of endolymph may be responsible for some of the symptoms of Méniére's disease. Patients with this malady often complain just before an attack of vertigo of pressure or headache on the side with impaired hearing. Some of the characteristics of Méniére's disease that may be due to endolymph changes are: the auditory and vestibular apparatus are usually both involved; the deafness is always of the inner ear type, but, unlike that due to a cochlear nerve or a destructive labyrinth lesion, the hearing may fluctuate as much as 50 dcb.; the attacks of vertigo are similar to the severe reactions produced with excessive stimulation of the normal labyrinth by turning or douching with hot or cold water. In Méniére's disease, the cause of the vertigo is an irritative and not a destructive lesion, since long remissions during which the patient has no vestibular symptoms are common. The vestibular response to

caloric and turning tests in these patients may be absent at one or more examinations and normal at a subsequent test. In other words, all symptoms of Ménière's disease, including the headache, tinnitus, deafness and vertigo, may fluctuate from time to time, indicating an irritative rather than a destructive lesion, and the absence of involvement of other cranial nerves or of the central nervous system suggests that the primary cause may lie in the labyrinthine fluid which bathes both the vestibular and the cochlear end-organs — the endolymph. It is certain to be a fruitful field for investigation.

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MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Oct. 8, 1937.

(Continued from July issue.)

Fundus Photography (lantern slides shown). Dr. M. F. Fellows.

It has always been the desire of the ophthalmologist to have as complete records and descriptions of pathologic changes of the retina as possible. Since the early days of photography, attempts have been made to photograph the retina, and the literature of the past few years is abundant with reference to this work.

In the April number of the American Journal of Ophthalmology, 1864, appeared an article by O. M. Rosebrugh, of Toronto, entitled, "On a New Instrument for Photographing the Fundus Oculi," and this was accompanied by a drawing of the camera used for this purpose. He had been preceded, however, by two years in attempting this process by Noyes, of New York, who appears to be the first to endeavor to photograph the retina. In 1907, Dimmer published his first atlas of retinal photographs, and in 1915 the process became much more practical when Nordenson attached photographic equipment to the Gulstrand ophthalmoscope. Since that time, much progress has been made in retinal photography, until now the practical application of color photography holds out much of promise.

At the time I was on my fellowship at the University of Minnesota, in 1930, Dr. Burch installed in the department a Zeiss reflex-free fundus camera. The advantage of this method of recording in case study was very apparent. Over a year ago, I was fortunate in being able to obtain a fundus camera for my private use, which camera, although it is the old type and not reflex-free, serves the purpose very admirably.

The use of the camera lies not only in taking individual photographs of the retina for record purposes, but also in taking several pictures so that a pathologic process may be studied over a period of time and changes from time to time noted. It may be used to show definite vascular changes in hypertensive fundi from time to time, the changes in choroiditis, tumors and hemorrhages, or the changes in operative conditions such as retinal detachments. It may also be used as record of intraocular injuries. One advantage is that the photograph may be studied in detail long after the patient has left the office. A photograph will never displace a thorough and comprehensive ophthalmoscopic examination of the fundus and must necessarily be considered as supplementary. Changes in tumors, etc., may be actually measured in the photograph and any progress or regression in size duly noted.

This evening, I am presenting four series of fundus photographs, each of somewhat unusual interest to me and ones which I believe, will illustrate the value of a fundus photograph over a written description.

The first picture is that of a thrombosis of one of the peripheral branches of the retinal artery. This patient was an interne, age 30 years, whose only complaint was that, when looking through the microscope with his left eye, in the past three or four months he had noticed spots, which he said appeared to be about the size of red blood corpuscles. This picture, taken Feb. 6, 1937, reveals a white linear streak throughout a section of one of the arteries and,

peripheral to this, many small hemorrhages. This is in the extreme temporal periphery of the fundus. The next picture was taken April 29, 1937, and shows the circulation re-established in this vessel, and at the proximal end of the thrombosed area you will note a marked tortuosity for a short distance, which to me represents the establishment of a new channel around an obstruction. This man was hospitalized upon two occasions and a complete physical examination, including X-ray and serology, performed without revealing any source of the trouble; however, it is interesting to note that on April 16, 1937, he was admitted with a phlebitis and lymphangitis of the left arm, following giving of blood for a transfusion. Whether or not this man is more subject to thrombus formation than the ordinary is something to conjure upon. The fact that the blood current here was re-established leads one to wonder how often this occurs without being brought to our attention.

The second case is that of a serous detachment of the retina, which reattached without treatment. This was a man, age 55 years, who awoke, Aug. 2, 1937, with a severe headache involving the entire left half of the head. Shortly after awakening he was seized with a very severe sneezing spell, following which he noticed that the vision in the left eye was blurred to the left and in the lower half of the field. The headache cleared up, but the vision remained about the same. He was seen by me Aug. 6, 1937, at which time the vision of the right eye was 20/15 and fundus examination normal. The vision of the left eye was such that he could read an occasional letter at the right end of the 20/25 line. The left pupil was slightly larger than the right and reacted more slowly to light. The fundus examination, as you see, revealed a roughly rectangular bluish area, slightly raised, and extending from the disc margin through the macula about four disc diameters temporally and about one disc diameter in width, with the retinal vessels slightly elevated over this area. The visual field showed a scotoma corresponding quite accurately to this area. The physical examination revealed nothing pathological. This man refused hospitalization and continued his usual routine. He returned Aug. 10, 1937, at which time the second picture was taken. You will note this shows a contracting of the borders of this entire area, the blood vessels are less elevated and there is a white patch at the margin of the disc, which appears as a patch of choroiditis and, I believe, is the source of the original trouble. The last photograph, taken Aug. 16, 1937, 10 days after the original, shows an almost entire reattachment of the retina. The scotoma upon both of these last visits showed a corresponding shrinkage in size. This man was from out of town and, unfortunately for record purposes, did not return subsequent to this last examination. However, I am sure that he would have returned had there been any recurrence of symptoms.

The next series of four photographs I present, not because they show anything particularly unusual, but only because they show the same fundus with hypertensive changes through four successive stages. This patient, a white woman, age 54 years, came to the office, Jan. 23, 1937, complaining of poor vision in the left eye for about the past month, with frequent sensations of pain and discomfort in the eye. About two years previous, she had an attack of redness and pain in this eye. At present, she had frequent afternoon headaches, which she described as being "all over the head." Examination revealed the vision in the left eye to be 10/200 and the fundus showed, as illustrated, a marked sclerosis of the arteries, with increased tortuosity of the smaller arterioles, depression of many of the arteriovenous crossings, and in the lower temporal quadrant large massive retinal hemorrhages with evidence of new blood vessel formation. Physical examination revealed her blood pressure to be 210/120, with negative urine, P.S.P. function was 58 per cent in the first hour, and 17 per cent in the second. A number of abscessed teeth were found and removed. Her family history revealed that two of her sisters had hypertension. The second photograph, taken March 30, 1937, shows partial absorption of the hemorrhages, and the macular region to be surrounded by a ring of white exudate, the new blood vessels being more prominent. The blood pressure was 180/100. The third photograph, taken April 20, 1937, shows further absorption of the hemorrhages, with a decrease in the amount of exudate. The blood pressure was 200/90. The vision remains the same, 10/200.

The last series of six photographs has been of great interest to me and is still an unsolved problem. I present these, not because I can give it a definite diagnosis, but because it shows in close series the development of a macular lesion from a very early stage. This patient was a girl, age 30 years, who had noticed, a year previously, that she was seeing double, that she could see two people approaching when only one was actually there. She thinks that she could see the entire object but that the person might have appeared taller. For perhaps the last six months she has noticed that the vision of the right eye was poorer and she now sees only the lower one-half of an object. She has had no pain or discomfort in the eye. Examination, July 27, 1937, revealed the vision of the right eye to be limited to large objects and movement only. Fundus examination revealed this small, slightly elevated, rounded area with a bluish color in the centre of the macula, with a very marked dark halo about the area and striae radiating in all directions from this central area. The retinal vessels are seen to pass over this area. The disc was undisturbed and the tension was normal. The second of this series was taken Aug. 6, 1937, nine days after the first, and shows a very marked increase in the size of the lesion, the margins becoming more sharply defined, the original elevated spot still prominent and the surrounding congestion the same. The one taken one week later, Aug. 13, 1937, shows a still more sharply defined area about two times the diameter of the disc, slightly elevated, about one and one-half diopters, with an apparent indentation in the centre of the area. The reaction about the lesion is as marked as before. The next picture, taken Sept. 3, 1937, six weeks following the first observation, shows the lesion to have become more oval, with the lateral margin pointed and the medial margin rounded. The subsequent photograph, Sept. 23, 1937, shows a darkening of the nasal border of the lesion, which has assumed a dull, hemorrhagic appearance. The size of the lesion is approximately the same, and the last photograph, taken on Sept. 30, 1937, gives the impression that the lesion is flattening out, the dark area is accentuated and the surrounding inflammatory changes in this, as in the last two, seem to show over a wider area than the earlier photographs. I have been uncertain whether this mass represents a sarcoma or a purely inflammatory lesion. At first, I was inclined to favor the diagnosis of sarcoma. The patient was seen by two consultants, both of whom took the view that it was probably inflammatory in nature. Should it begin to decrease in size, I would presume that diagnosis to be corroborated; however, I am still uncertain whether to continue observation or to suggest removal of the eye. For a time, at least, I plan on further observation. The physical examination includes spinal Wassermann and von Pirquet, and complete physical examinations were negative. Her history revealed an abortion about the time of onset of the original symptoms, and a Neisserian infection eight or 10 years ago.

These photographs do not show up as well on the slide as they do in the paper print. Should anyone care to examine them, I have the photographs here, as well as a series of stereoscopic views of the last macular lesion.

JOINT MEETING OF

THE NEW YORK ACADEMY OF MEDICINE

SECTION ON OTO-LARYNGOLOGY.

AND

THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.

SECTION ON OTO-LARYNGOLOGY.

Held at the New York Academy of Medicine, March 16, 1938.

Modification of the Semilunar Ganglion Approach Used in Surgery of the Petrous Pyramid. Dr. Marvin F. Jones.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. OSCAR V. BATSON: This is a very interesting approach to the medial portion of the temporal bone. I think that it is a practical approach because it is under such complete ocular control. In reviewing this approach I shall remind you of the essential shape of the petrous part of the temporal bone. It is a pyramid with the base externally and the apex toward the midline, and somewhat anterior. The pyramid can be considered as having four sides. One side is in the middle fossa, one in the posterior fossa, and a very rough jagged side of the pyramid is on the base of the skull. If you will use your imagination, there is a fourth anterior side that consists of part of the glenoid cavity, part of the bony canal wall and part of the tympanum. Toward the midline or anteromedial, as Dr. Jones puts it, is the portion which contains the carotid artery and just beyond that, of course, is the sphenoidal sinus and the cavernous venous sinus. This apical part of the bone may be pneumatic, diploic or sclerotic. I do not believe the sclerotic type is very often involved in infection. The pneumatic type is probably involved as often as the mastoid process itself. The diploic may be involved, but usually late in the course of an infection.

The principal obstacle to the surgical approach through the temporal bone is the labyrinth itself. In the case of a dead labyrinth, the translabyrinthine approach appears to be the most practical and the most obvious; this approach gives you immediately access to the petrous apex. With a live labyrinth I think we may classify the proposed surgical approaches under four headings: First, the retrolabyrinthine. I do not believe this is very practical, because we encounter there the saccus endolymphaticus, the upper portion of the sigmoid part of the transverse sinus, a portion of the porus acusticus internus, and perhaps the petrosal sinus. Second, the inferior or hypolabyrinthine approach. This has been investigated by Dr. Meyerson of this group and I believe it is particularly suited for establishing simple drainage, but is not adequate for exposure under observation. There are so many structures on the base of the skull that would have to be severed to obtain adequate inspection. The third is the prelabyrinthine approach. This has been explored by Kopetzky, Almour and Lempert. By removing the anterior canal wall and by exposing the carotid artery, it is possible to reach the apical portion of the bone. Those approaches to the petrous apex all require a profound knowledge and a steady hand. The relations are very narrow, but the results seem to be excellent. The fourth approach is epilabyrinthine. It can be carried out in a very pneumatic bone by going over the labyrinth, but such extreme pneumaticity is rare. This leaves for consideration the approach Dr. Jones presented tonight, the trigeminal ganglion operation approach. Dr. Jones has

covered well the difficulties in this epilabyrinthine approach. Of course, it can be made by craniectomy as well as by craniotomy. This exposure gives you a clear view. There are the difficulties that he mentioned, particularly that of the adherent dura at the petrous apex, and the presence of the middle meningeal artery which must be elevated or sometimes interrupted. The greater superficial petrosal nerve is in the field. I would like to add that in children particularly, the geniculate ganglion does not lie covered with bone, but is in the middle cranial fossa covered only by dura; therefore, additional caution must be observed in these cases.

The principal difficulty in the approach would be that the average oto-laryngologist is not particularly familiar with the routine methods of the brain surgeon. If that familiarity is possessed or if it be obtained, then the procedure may be ideal, because, with the brain retracted and with lighted retractors holding the dura containing brain upward, the whole apex lies exposed. I think the principal arugment in favor of this approach, in addition to the argument that you are working through an unrestricted field, is that it is possible by this means to avoid a radical operation with its resultant hearing loss.

Parapharyngeal Hemorrhage: Diagnosis and Treatment. Dr. Francis W. White and Dr. Louis Hubert.

In presenting their case histories and incidental remarks, the authors realize that much has already been written on the subject. The fact remains, however, that comparatively many deaths occurred, the number of which might have been lessened if there had been a better understanding of the diagnosis and treatment.

Six case histories are presented, four males and two females, the youngest patient of the series being 5½ years of age, and the eldest, 60 years of age. Concomitant conditions especially were scarlet fever, diabetes mellitus, one tooth infection and extraction. Four required operative procedures, and two recovered under conservative treatment.

The anatomy of the area is not dealt with in detail, as this has already been covered thoroughly by others. Special stress, however, is laid upon the relationships and contiguity of the IXth, Xth and XIIth cranial nerves, the cervical sympathetic trunk and its superior ganglion, the means of communication between the parotid space and the pharyngomaxillary space by means of a gap in the fascia, and also the presence of the facial nerve in the parotid space.

An analysis of the case reports of parapharyngeal infection submitted suggests the following:

1. The degree of infectivity of the causative organism for a given person determines the seriousness of the parapharyngeal invasion. 2. Conservative treatment of parapharyngeal infection is in many instances successful. 3. Free, active hemorrhage from the throat, with or without apparent throat inflammation, requires not local treatment, but bold, external operative measures. 4. Meddlesome incision of a lateral pharyngeal swelling may lead to hemorrhage. If left alone, and treated conservatively, ligation may not be necessary. 5. Aural hemorrhage, associated with parapharyngeal infection, requires immediate ligation of the common carotid artery. 6. If any doubt exists regarding erosion of the internal carotid artery, the external carotid and the ascending pharyngeal arteries should be ligated, and a loose ligature placed around the common carotid artery. 7. Ligation of the common carotid artery, when done slowly—minutes, not seconds—and an attempt is made to actually prevent the flow of blood beyond the ligature, not necessarily to feel the severance of the intima, is a life-saving operation, as compared to temporizing methods by the intrapharyngeal route. 8. Throat and ear hemorrhages associated with trismus, involvement of nerves, as noted above, lateral pharyngeal tumefaction, sepsis, swelling and tenderness of the corresponding side of the neck, indicate pharyngomaxillary fossa infection.

DISCUSSION.

DR. WARREN B. DAVIS: The papers by Dr. Hubert and Dr. White have been both interesting and instructive, from anatomical, pathological and clinical viewpoints.

In discussing these papers, I wish to state that I am doing so as one who has not had hemorrhage from vessels in this area secondary to infections, although I have had my full share of infections in these areas due not only to otolaryngologic conditions, but also to those encountered in maxillofacial surgery. The infections which I have had in the parapharyngeal area without hemorrhages occurring have been those that were secondary to peritonsillar abscess, retropharyngeal abscess—both from the breaking down of lymphnodes and those secondary to mastoid involvement, parotid abscess, those infections of dental origin due to peridental infections, necrosis of the mandible and compound fractures of the mandible, submaxillary abscess due to calculi in the ducts or the gland, and from Ludwig's angina with extension laterally and posteriorly, as well as infections from the sublingual region.

It is interesting to note in medical literature that this subject, as far as infection goes, has been under discussion for 102 years, since the paper of Ludwig of Stuttgart in 1836. In our own country it seems to have taken on additional clinical interest since the studies of Dr. Mosher, published in his several papers starting about 1920. Besides the papers that were quoted by Dr. White, I feel that it might be justifiable to refer to two or three other papers which give some additional information. The frequency with which these hemorrhages have occurred in some clinics makes me more appreciative of the faithfulness of my guardian angel in seeing that they have not occurred in any of my cases. In a paper by Goodyear of Cincinnati, in 1929, he reported eight fatalities which had occurred in the Cincinnati hospitals during that year from hemorrhages secondary to peritonsillar abscesses. Goodyear also reported statistics from Newcomb's paper, from which he had reported 41 peritonsillar abscesses followed by hemorrhage, and of these 41 cases there were 23 deaths. Other fatalities have been reported by Carmody, King, Langley, and others in smaller numbers. In 1936, Dr. Baker, of New York, read a paper before this Academy on the subject of throat infections and hemorrhages. At the meeting of the American Rhinological and Otological Association at Atlantic City in 1937, Porter reported some cases of infection with hemorrhage and thrombosis. In April, 1937, Boemer, of St. Louis, reported a study of 75 cases of deep pus in the neck in which there were two deaths, one from a neglected mastoid, and the other from hemorrhage in a neglected peritonsillar abscess which was not recognized until quite late. It is of interest that Wishart reported 41 cases of retropharyngeal abscess from the Toronto Children's Hospital, all of which were operated on from within the mouth, and all recovered.

I have enjoyed these papers immensely and I think the lesson that they carry to us is that all of us must be prepared to act promptly and efficiently when the emergency arises. To do that we must not only keep close clinical observations, but we must thoroughly know the anatomy of these regions, both as regards the triangles formed by the muscles and also the relations of the bony structures, since swelling may obliterate the muscular landmarks. Those who have seen the beautiful dissections by Dr. Batson, showing the blood vessels of the head and neck, appreciate the very numerous anatomoses between the vessels of this area, and the consequent difficulties in completely controlling bleeding by a single ligation. I would like to ask Dr. White how completely ligation of the carotid, in his cases, has controlled the bleeding and whether or not there has been much bleeding from above or whether it has been minimized by the thrombosis.

DR. FRANCIS W. WHITE: Inasmuch as we had no deaths in our series of patients, and consequently, no postmortem examinations, it would be hard to answer Dr. Davis' question, regarding postligation hemorrhages. The stress laid upon doing the ligation of the common carotid artery slowly is to lessen the chances of intracranial damage. Two of our patients suffered from hemorrhages after ligation of the common carotid artery, in one patient as late as the eleventh day. These experiences are worrysome, but apparently the condition adjusts itself. Of course, it must be due to collateral circulation. When opportunity offers, this will be an interesting subject for investigation.

(To be continued in a succeeding issue.)

BOOK REVIEW.

Sound Waves; Their Shape and Speed. By Dayton Clarence Miller, D.Sc., D.End., LL.D.; Professor Physics, Case School of Applied Sciences. 164 pages with 64 illustrations, bibliography and index. New York: The Macmillan Co., 1937. Price, $2.75.

The lectures on "Sound Analysis" delivered by Dayton C. Miller at the Lowell Institute were collected in book form in 1916 and published under the title, "The Science of Musical Sounds." This book has been quite widely accepted as a classic in this field. Dr. Miller's later investigations of sound now appear in a new volume, "Sound Waves; Their Shape and Speed."

The first part of "Sound Waves" may be said to be a continuation of "The Science of Musical Sounds." The phonodeik, which was used so extensively in the first series of studies, is described in greater detail in this new volume, but the mathematical analysis of the wave forms of the vowel sounds is not discussed.

Dr. Miller, in 1919, became interested in the study of "the air disturbances produced by bullets from the army service rifle." This investigation necessitated the use of the electric-spark method of photography. Chapter IV contains a resumé of the various devices used for spark photography and many illustrations showing the form of air waves produced by bullets of various shapes. This part of his study may be said to be in preparation for the investigations described in the later chapters.

In co-operation with the Ordnance Department of the U. S. Army, Dr. Miller went to the Sandy Hook proving ground to study the "pressure developed in the sound waves produced by the discharge of large guns, and for the determination of the velocity of the explosive sounds and of the normal velocity of sound in free air." It is perhaps these studies of air pressures which should be the most interesting to otologists, as well as to army surgeons.

The measurement of these air pressures was determined by means of an instrument devised by Miller and called the baroscope. With it, the pressures on the ground 12 feet in front of the muzzle of a 12-inch rifle were found to be 542 pounds per square inch, and at the breech the pressure was five pounds per square inch. The phonodeik, which was also used in this investigation, showed that the explosive sound produced "no true vibratory wave form; instead, there was a positive pulse (compression), rising rather abruptly to a maximum, and falling to a negative pressure (rarefaction).

The physiological effects of these pressures were studied by Dr. D. R. Hooker and are discussed by him elsewhere (Amer. Jour. Physiol., 67:219, 1924). Miller's quotation from this study includes this statement, "Exposure of animals, 10 to 20 feet in front of 10-inch and 12-inch rifles, yielding a concussion pressure of 18 to 19 atmospheres, usually produced primary shock. This condition is essentially instantaneous in onset. . . . The concussion pressure adequate to produce shock results in extensive laceration of the tympanic membrane of the ears."

The last chapter is devoted to a study of the velocity of the sound of these explosions in free air. In this determination, Miller finds himself in agreement with the value reported at the International Congress of Physics at Paris in 1910, viz., 331.36 metres per second.

"Sound Waves" is written in Dr. Miller's characteristic style. The absence of mathematics makes it easily understood by those untrained in higher physics and mathematics. It will, undoubtedly, be received by students on the same basis as "The Science of Musical Sounds." C. C. B.